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# Indications for Total Pneumonectomy*

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The operation of total pneumonectomy is a comparatively new procedure. The first successful one stage removal of an entire lung was accomplished ten years ago last April.[1] The operation was performed because of a squamous cell bronchiogenic carcinoma. The patient, a physician, is living and well and is conducting a very active practice. Two years previously, however, Nissen[2] in 1931 caused the entire left lung to slough away in a child with bronchiectasis by applying a ligature around the hilum. In 1932 Haight,[3] using a similar procedure, in a two stage operation produced a slough of the entire left lung in a child with bronchiectasis.

The operation of total pneumonectomy in one stage has now been performed many times by many surgeons. During the ten and one-half years of its existence various technics have been employed out of which certain standard principles have developed. Rienhoff[4] early demonstrated by his first case, in which the operation was carried out at almost the same time as my first case, that it is not necessary to perform a thoracoplasty in order to obliterate the pleural space and that individual ligation of the hilar structures with a careful suturing of the bronchus has many advantages over the mass ligation of the hilar pedicle. These principles have now been generally accepted as procedures of choice. Total pneumonectomy, therefore, today usually means an operation for the complete removal of a lung, performed in one stage and with individual ligation of the hilar vessels and suturing of the bronchus. Occasionally, however, because of exceptional technical difficulties mass ligation of the hilum is still practiced and in rare instances a two stage operation is carried out.

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The fact that an entire lung can now be removed gives rise immediately to the question of what are the indications for the operation. Obviously the conditions for which such a procedure would be recommended would be those in which one lung is so seriously diseased that its retention would cause the death of the individual or interfere to a marked degree with his well being. These conditions fall naturally into three main groups, (a) primary new growths, (b) chronic suppurative processes involving most if not all of one lung, and (c) certain selected cases of pulmonary tuberculosis.

Bronchiogenic carcinoma constitutes at the present time the chief indication for the operation of total pneumonectomy. This condition, formerly considered to be rare, is now known to constitute perhaps from 8 to 10 per cent of all carcinomas. It is so common that any male patient of middle age or beyond who develops a chronic cough with the expectoration of sputum which does not contain tubercle bacilli should be considered to have it unless there is positive evidence to the contrary. The only treatment likely to be successful that has been demonstrated up to the present time is surgical removal. Any reported successful results from radiation therapy are to be regarded with suspicion.* It is theoretically possible that in certain early and favorable cases a limited resection of only a portion of the lung (as for example, a lobectomy) may be sufficient. Yet it has become a standard practice in surgery to sacrifice the entire organ which is the site of malignant disease rather than to perform any less radical operation, unless the removal of the whole organ involved is incompatible with life. This practice has been founded on much trial and error during the development of the modern surgical treatment of malignant disease. As a matter of principle, therefore, it would seem wiser to perform a total removal of the lung rather than the removal of only a limited portion.

A bronchoscopic biopsy showing the presence of a carcinoma is, therefore, generally to be regarded as an indication for total pneumonectomy. Certain findings, however, should usually be considered as constituting contraindications because their presence is evidence of such widespread extension of the growth that complete removal of the cancer can hardly be considered possible. These findings are:

1) The presence of bloody pleural fluid. Such a finding is almost certain evidence that at least the visceral pleura and probably also the parietal pleura has been invaded by the growth. When a pleural

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*In one case with which I am familiar Chevalier L. Jackson removed a small early carcinoma through a bronchoscope. I have examined the specimen and am willing to state unhesitatingly that it represents a carcinoma. Moreover, when I saw the patient two years later there was no evidence of any recurrence either on bronchoscopy or otherwise. Such a case, however, should probably be regarded as most exceptional.

effusion of clear fluid is found it is nearly certain that at least the visceral pleura has been invaded, although of course it is theoretically possible that the effusion has formed as a response to some other etiological factor. Actually in the few cases in which I have performed a total pneumonectomy for carcinoma in the presence of a pleural effusion the patient has invariably died within a year, of recurrence of the trouble.

2) Paralysis of the corresponding half of the diaphragm. This finding, readily determined by a fluoroscopic examination, is evidence of an invasion of the phrenic nerve and, in my experience, precludes a successful operation.

3) Paralysis of the left vocal cord in cases of left-sided bronchiogenic carcinoma. When this finding is present it usually denotes an invasion of the left recurrent laryngeal nerve as it passes under the arch of the aorta, and it indicates that the growth is already so extensive as to preclude a successful operation. Exceptionally, however, the paralysis of the vocal cord may have a different explanation. For example, I have had a recent experience in which I declined to operate on a man because of this finding. But a few weeks later the cord had begun to move again, a fact which suggested that probably its paralysis had not been due to invasion of the nerve. A total pneumonectomy was then successfully carried out with a reasonably good prospect of ultimate cure.

4) Severe pain in the thoracic wall or down the arm. This is a bad sign and generally is evidence of an involvement of intercostal nerves or of the brachial plexus. The presence of a moderate amount of pain, however, should not by itself preclude an exploratory operation to investigate the possibility of a successful pneumonectomy.

5) Bronchoscopic evidence of extension of the tumor into the trachea will usually contraindicate pneumonectomy although occasionally it is possible to remove even a part of the wall of the trachea. Likewise firm fixation of the hilum as noted on bronchoscopic examination is an unfavorable sign but does not always indicate that pneumonectomy cannot be accomplished.

6) The presence of distant metastases as a rule will contraindicate the removal of the lung, although in exceptional instances it may prove justifiable to remove both the lung and a solitary metastasis.

7) The question of the age of the patient is important from the standpoint of operability, but at least up to the present time there is no fixed limit. The general condition, and particularly that of the cardiovascular apparatus is more important than the chronological age. My oldest patient to survive the operation was 68. Another patient of 70 years made an immediately satisfactory recovery but died suddenly of coronary thrombosis one week later.

One question of great importance regarding the use of the operation of total pneumonectomy for new growths concerns the best method of treating that group of tumors usually termed bronchial adenoma. The controversy over this matter is due chiefly to a difference of opinion as to the nature and potentialities of this tumor. By many it is regarded as an essentially benign condition for which often only a limited resection of lung is indicated. Womack and I,[5,6,7] however, regard it as a neoplasm which is always potentially malignant and very often malignant at the time of its first clinical recognition. For that reason, therefore, also because it is usually located in one of the larger bronchi, I consider that the safest method of treatment in most cases is total pneumonectomy. In the limited scope of this paper it would not be possible, nor would it be appropriate, to attempt to present convincingly the argument favoring the viewpoint of the essential malignancy of this tumor. Those who are interested may read our various papers on this subject. For the purposes of this paper it will perhaps be sufficient to state that when it assumes its malignant features it develops a marked tendency to invade neighboring structures and later to develop metastases in regional lymphatic glands and even in distant organs. In our opinion this tumor is the origin of most of those carcinomas, other than the squamous cell variety, which carry the common names of "oat cell carcinoma," "small round cell carcinoma," "alveolar carcinoma" and many if not all of the bronchial carcinomas designated as "adenocarcinoma." A common feature of these tumors is their pleomorphism. Sections taken from different areas may present such varied appearances of structure that almost all the common names used by the orthodox pathologists could be applied to the same tumor depending upon what area of it was being examined. Cartilage and even bone are sometimes included. Because of their structural characteristics and their tendency to invade neighboring tissues we have recognized many similarities between these tumors and the common ones of the salivary glands. Accordingly, we have preferred to call them mixed tumors of the bronchus instead of using the more conventional term of bronchial adenoma. We feel that our name is more expressive of the embryonic origin and of the most important characteristics of the tumor.

Sometimes total pneumonectomy will be required in cases in which the less common tumors of the lung are present. These will comprise cases of sarcoma as well as certain instances of benign tumors in which it will be difficult to save any lung tissue because of either the extreme size or the location of the tumor.

Any discussion of the indications for total pneumonectomy in bronchiogenic carcinoma is incomplete without some reference to the management of those cases in which, although suspected, the

presence of the condition has not been established with certainty. A positive diagnosis can be made before operation best by bronchoscopic visualization of the lesion, supported by a positive biopsy. Although others have occasionally been successful in making a diagnosis by use of a punch biopsy through the chest wall, we have not found the procedure helpful. In those cases in which the growth has arisen in one of the smaller bronchi it is generally impossible to visualize the tumor through the bronchoscope. This is particularly true when the lesion is in an upper lobe. In our own experience it is possible to establish a positive diagnosis by bronchoscopic biopsy in only about 75 per cent of cases. The problem of what advice to give to the 25 per cent without a positive diagnosis is an important one. The answer, in my opinion, is generally fairly simple. If there is a reasonable suspicion of the presence of a carcinoma, an exploratory thoracotomy should be performed without delay; for in that way many lives will be saved. One of the most disheartening experiences which a thoracic surgeon has is seeing the large number of patients with hopelessly inoperable cancers. Too often the advice of the physician has been to wait to see if anything happens. The only gainer by this delay has been Death, who has claimed another victim.

The clinical evidence, after all, is fairly trustworthy if the chief features are present. A chronic cough with the expectoration of blood-streaked sputum in which tubercle bacilli are not found, together with a hilar infiltrative lesion seen by x-ray, almost certainly indicate the presence of a bronchiogenic carcinoma, especially if the patient is a male of middle age or more. These features are sufficient to justify an exploratory operation regardless of the failure to see a tumor on bronchoscopic examination or of a failure to find a bronchial obstruction with lipiodol. In twelve cases of this kind in our experience pneumonectomy was performed and in eleven of the twelve carcinoma was found. In the other case the lung was removed although subsequent examination of it showed only the presence of a chronic inflammatory process.

Chronic suppurative disease involving all lobes of one lung is best treated by total pneumonectomy if the other lung is free from disease. Such a condition is found particularly in the cases of so-called unilateral universal bronchiectasis and in suppurative unilateral congenital cystic disease involving all of one lung. There is good reason to believe that in many instances the condition sometimes termed congenital bronchiectasis represents merely a stage of infection of congenital cystic disease. The cysts are always liable to infection and the best evidence at hand indicates that when once they become infected there is little or no chance of their becoming healed. Generally, therefore, the presence of congenital cystic dis-

ease involving all the lobes of one lung is an indication for pneumonectomy regardless of whether or not infection has already become established. Indeed it is preferable and safer to perform the operation before infection has set in. The condition is most commonly recognized in children and in young adults. The fact that it is seldom found in patients who have reached middle age is very significant, in my opinion. Does it not suggest that the child with congenital cystic disease has a poor chance to reach middle age because of the infection which is almost inevitable? Is not this observation in itself an argument favoring the early removal of a cystic lung? There is another reason for removing this malformed lung at an early age. Womack and I[6,7] have shown that there is a high incidence of malignant tumor formation in cases of cystic disease. This finding is in line with the well known and commonly accepted fact that malformations have a tendency to undergo malignant changes. A striking example is the high incidence of malignancy in cases of teratoma.

There are other cases of non-tuberculous infection of the lung in which total pneumonectomy is indicated. Certain cases of nonspecific chronic or recurring unilateral pneumonitis associated with a considerable degree of fibrosis respond very favorably to the removal of the lung. Also a patient with the rare condition of lipoid pneumonia, when it is confined to one lung, may be relieved of an otherwise hopeless disease by the operation of total pneumonectomy. I have had one successful case of that sort. It is probable that some cases of fungus infection will be successfully treated by total pneumonectomy as time goes on.

We come now to a consideration of the place of total pneumonectomy in the treatment of tuberculosis. Even as far back as the latter part of the last century the idea was frequently expressed that perhaps at some time it would be possible to remove a part or even the whole lung that is diseased with tuberculosis. In fact, some of the earliest attempts of intrathoracic surgery were made for the purpose of removing parts of the lung because of this disease. Some of these were successful. Operations for this purpose, however, fell into disrepute because of the prohibitive mortality and the generally unsatisfactory results. Within recent years they have been revived again somewhat cautiously. The report of Dolley and Jones[8] in 1940 at the meeting of the American Association for Thoracic Surgery, together with the discussion which followed it, indicated that there is certainly a definite place for lobectomy in certain cases and probably a place for total pneumonectomy in a very small group of cases. The cases in which one may consider the operation of total pneumonectomy indicated are those in which the main bronchus of the diseased lung contains a stricture which prevents adequate aeration

and drainage of secretions, cases of persistent thick-walled and uncollapsible cavities and those in which a universal tuberculous bronchiectasis exists. Probably only a very small number of patients with these conditions will be suitable for the operation and for the most part they will be those in whom a previously performed thoracoplasty has failed to give the desired results.

No discussion of the indications for a relatively new operation is complete without some idea of the risks involved. As regards the operation of total pneumonectomy I shall present here only the experience of Blades and myself.

We have had a series of 104 cases of total pneumonectomy. Of these there have been 81 operations for neoplasm and 23 operations for other conditions. This represents a period of approximately ten and one-half years. In the early years of the development of the operation the technic was often faulty and the indications were not so sharply drawn as at present. I prefer, therefore, to concentrate on the results obtained since January 1, 1939, a period of nearly five years. During that time we have performed 75 total pneumonectomies of which 70 were for malignant neoplasm. In this group there were 21 hospital deaths, a mortality therefore of 30 per cent. Mortality figures expressed in this manner, however, are not so significant as they might seem. For example, in our last 25 cases for neoplasm there have been only three deaths, or a mortality of 12 per cent. One of these deaths was in the man, previously mentioned, 70 years of age, who died a week after operation in an attack of coronary thrombosis. I was led to operate on him, despite his age, because of the remarkably uneventful convalescence which the patient, 68 years of age, also previously mentioned, had had shortly before this operation. If one wishes to select his cases more carefully, the operative mortality will be still lower and may approximate zero, but he will also deprive many patients of relief from their trouble. This is the dilemma in which the thoracic surgeon finds himself. Shall he offer a chance to the bad risk victim of bronchiogenic carcinoma even if it is only a small one, to be cured of his otherwise hopeless condition, or shall he decline to operate because of the great danger of an operative mortality? I think most of us will offer the patient a chance of recovery. After all, an operative mortality of 12 per cent in 25 cases of malignant bronchiogenic tumor is a thoroughly respectable one, and should dispel the all too common idea that the operation of total pneumonectomy for bronchiogenic carcinoma carries with it an enormous operative risk. It should be remembered that without operation the risk is 100 per cent.

Interest in the results of total pneumonectomy for bronchiogenic carcinoma, of course, should not be confined merely to operative

mortalities. Questions that naturally arise are, Is there a chance of a permanent cure? Will the patient be permanently crippled?

Let us answer the last question first. There is no impairment of function or usefulness for the ordinary person of middle age. It is possible that a professional wrestler or boxer might not be able to carry on his occupation, but there is no restriction of ordinary activities or even of such indulgence in athletics as golf, hunting, fishing, horseback riding, etc.

In reply to the first question, I may say that I now have five patients who are well more than five years after their operation and one for more than four years. Three of these patients had squamous cell carcinomas and the other three had carcinomas of other types. The periods of survival are as follows: 10 years and 7 months; 8 years; 6 years and 7 months; 6 years and 3 months; 5 years; 4 years and 1 month.


## RESUMEN

Desde cuando se ejecutó la primera neumonectomía total en una etapa con buen éxito en un caso de carcinoma broncógeno, en el mes de Abril de 1933, la operación ha logrado alcanzar una posición reconocida en el repertorio quirúrgico. Hasta la fecha las indicaciones principales son: (a) neoplasias primarias, (b) procesos supurados crónicos que afectan la mayor parte de un pulmón o todo un pulmón, (c) ciertos casos seleccionados de tuberculosis pulmonar. Hasta el presente el único tratamiento satisfactorio del carcinoma broncógeno es la neumonectomía total.

Hemos ejecutado la neumonectomía total en 104 casos en el Hospital Barnes. De éstos fueron hechas 81 operaciones de neoplasias y 23 operaciones por otras causas. En los primeros años de la evolución de esta operación la técnica era a menudo defectuosa y las indicaciones no estaban tan bien definidas como en el presente. Por consiguiente, prefiero concentrar la atención sobre los resultados obtenidos desde el 1° de Enero de 1939. Durante este período hemos ejecutado 75 neumonectomías totales, de las cuales 70 fueron por neoplasia maligna. En este grupo hubo 21 muertes en el hospital, una mortalidad, por consiguiente, del 30 por ciento. En nuestros últimos 25 casos, sin embargo, han acaecido sólo 3 muertes, o una mortalidad del 12 por ciento. Tenemos actualmente cinco pacientes que se encuentran bien desde hace más de cinco años después de su operación por carcinoma broncógeno y uno que ha estado bien por más de cuatro años. Tres de estos pacientes tenían carcinomas célulloescamosos y los otros tres tenían carcinomas de otros tipos. Los períodos transcurridos de sobrevivencia son los siguientes: Diez años y siete meses; ocho años; seis años y siete meses; seis años y tres meses; cinco años; cuatro años y un mes.


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# Collapse Therapy in Bilateral Pulmonary Tuberculosis*

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It is estimated that eighty per cent of the patients admitted to most sanatoria throughout the country have far-advanced pulmonary tuberculosis. The majority of our patients fall in this category.

To bring about arrest of disease in far-advanced bilateral cases, it is often necessary to employ some radical procedures of collapse therapy. The phthisiotherapist and the thoracic surgeon should work hand in hand. It is important that they should be able to evaluate the different operative procedures, and to employ them in such succession or combination as will be best suited to the condition of the particular patient.

The problem of obtaining and maintaining arrest of disease in far-advanced pulmonary tuberculosis often seems very discouraging, but a proper selection of cases and adequate measures often yield very satisfactory results.

In addition to a careful selection of cases, it is essential that special consideration be given to the physiology of respiration and its relation to the cardiovascular system. Frequent recording of the patient's vital capacity is extremely important and helpful.

Despite the progress made during the past decade by the surgical collapse of the tuberculous lung in unilateral cases, very little has been accomplished for the treatment of bilateral cases. Two reasons seem to account for this. One is a tendency of the phthisiotherapist to treat patients inadequately because of his adoption of a particular method of therapy. The other is a lack of team work between the phthisiotherapist and the thoracic surgeon, the former clinging to his medical measures of treatment, while the latter is prone to be surgically minded.

We are of the opinion that there are definite indications for each of the modern methods of collapse therapy as well as indications for their use in various combinations. It must be remembered that bilateral pulmonary tuberculosis is such a desperate condition that the important consideration is not the mortality rate, but whether

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the number of patients saved can justify our different procedures, radical as they might appear to some.

After careful selection, our patients having far-advanced bilateral tuberculosis are submitted to the simpler and more conservative measures of collapse therapy. These failing, the more radical procedures are employed. Hence, this type of patient is considered at the onset as a candidate for bilateral pneumothorax. We believe that in the vast majority of cases the patient should be given the advantage of this procedure.

The difficulties encountered in the induction and maintenance of bilateral pneumothorax are well known.

When pneumothorax is impossible, extrapleural pneumothorax or thoracoplasty in several stages should be performed bilaterally or in combination. Our results with oleothorax therapy have been rather discouraging, and we have more or less discarded this procedure.

Phrenic nerve surgery should be employed in cases of basal lesions complicated by lesions of the upper lobe of the contralateral lung.

The procedures of pneumothorax, thoracoplasty and phrenic nerve surgery are carried out with the same technic as for unilateral cases. We have omitted a detailed description of these operations, as this can readily be obtained in any textbook on thoracic surgery.

The following cases are presented to illustrate the different types of bilateral collapse therapy and the various combinations which can be employed.

## BILATERAL INTRAPLEURAL PNEUMOTHORAX

*Case I.*—C. A., white female, aged 26 years, whose father had died of pulmonary tuberculosis. The past history was negative.

The patient was admitted with an eight months' history of cough, expectoration, weakness, night sweats, loss of appetite and 18 pounds in weight. The temperature ranged from 97 to 99.6° F.; pulse, 80 to 90; respirations, 20 to 24. The sputum contained tubercle bacilli; sedimentation rate of 16 diagonal line (Cutler method); blood picture within normal limits.

Examination of the chest revealed moderate dullness over both upper lobes with cavernous breath sounds and medium rales (Fig. 1). Intrapleural pneumothorax was instituted on the right side and a pneumonolysis performed. Three months later, because of a persistent positive sputum and disease in the left lung an intrapleural pneumothorax was begun on this side, and later a pneumonolysis was performed (Fig. 2). The patient showed considerable improvement. The sputum became negative and the sedimentation rate dropped to 6 horizontal line. This patient is at home and well.

## INTRAPLEURAL PNEUMOTHORAX AND PHRENICLASIS

*Case II.*—A. G., female, white, aged 31 years. Family history and past history were negative. The patient was admitted from a general hospital where an emergency intrapleural pneumothorax was instituted for hemoptyses.

On admission the patient complained of cough, expectoration of 2 oz. of yellowish sputum daily, weakness and night sweats.

The temperature ranged from 97 to 102° F.; pulse, 90 to 110; respirations, 20 to 28. Sputum, positive; sedimentation rate, 19 diagonal line; blood picture within normal limits.

Examination of the chest revealed signs of pneumothorax on the left side. Moderate dullness was obtained over the lower half of the right lung, bronchial breath sounds and crepitant rales were heard in this area. When sufficient collapse of the left lung was brought about a pneumonolysis was performed (Fig. 3). Fourteen months later because of a persistently positive sputum and disease of the right base a phreniclasis was accomplished (Fig. 4). Following this procedure the patient showed con-

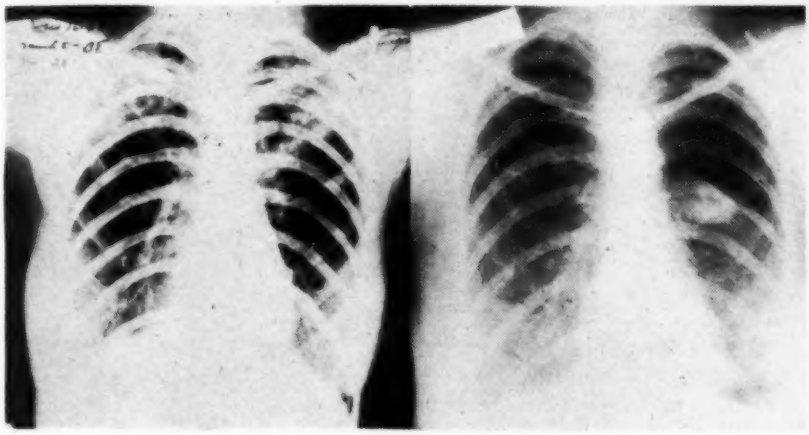

*Fig. 1* *Case I* *Fig. 2*

*Fig. 1*—Bilateral infiltration and bronchopneumonic reaction involving the upper third of both lung fields. Multiple small cavities in both infraclavicular regions. *Fig. 2*—Bilateral partial pneumothorax.

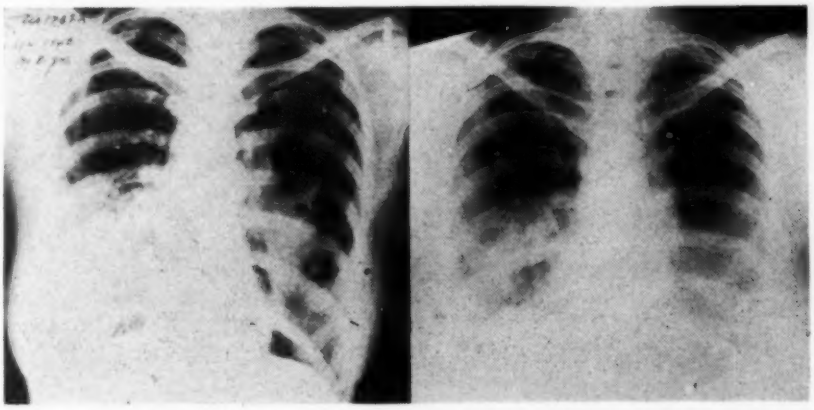

*Fig. 3* *Case II* *Fig. 4*

*Fig. 3*—Partial pneumothorax of the left lung with a large uncollapsed cavity and numerous adhesions. Infiltration with marked bronchopneumonic reaction involving the lower half of the right lung. *Fig. 4*—Partial pneumothorax of the left lung. Elevated right diaphragm secondary to phreniclasis. Area of dense fibrosis at the right base.

tinued signs of improvement both clinically and physically.

The sputum became negative and the sedimentation rate dropped to 11 diagonal line. This patient is at home and well.

## BILATERAL THORACOPLASTY

*Case III.*—W. D. was a white male, aged 30 years, whose family history and past history were negative.

The patient was admitted with a sixteen months' history of cough, expectoration of 3 oz. of sputum daily, weakness, loss of appetite and of fifteen pounds in weight. The temperature ranged from 97 to 99.4° F.; pulse, 80 to 96; respirations, 20 to 24. The sputum contained tubercle bacilli; sedimentation rate, 22 diagonal curve (Cutler method); blood picture within normal limits.

Examination of the chest revealed bilateral moderate apical dullness with cavernous breath sounds and medium rales. Artificial pneumothorax was attempted on both sides but was unsuccessful. A right-sided thoracoplasty was performed (Fig. 5). Following this operation he improved somewhat but his cough and expectoration persisted. Because of this and failure of the contralateral lung to improve, a left-sided thoracoplasty was performed (Fig. 6). His improvement after this operation was marked and his cough almost entirely disappeared.

The sputum became negative and the sedimentation rate dropped to 15 diagonal line.

The patient left the hospital against advice.

## THORACOPLASTY AND INTRAPLEURAL PNEUMOTHORAX

*Case IV.*—M. C., white male, aged 22 years. Mother and one brother had died of pulmonary tuberculosis. The past history was negative.

The patient was admitted with a ten months' history of cough, expectoration of 2 oz. of sputum daily, several hemoptyses ranging from 1 oz. to 4 oz., weakness, night sweats, loss of appetite and of 20 pounds in weight. The temperature ranged from 97 to 98.6° F.; pulse, 96 to 110;

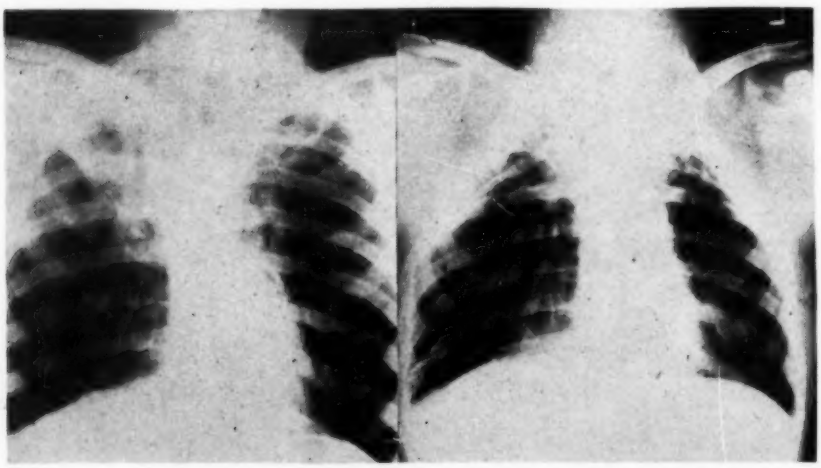

*Fig. 5* *Case III* *Fig. 6*

*Fig. 5*—Atelectasis and cavitation involving the right upper lobe. Infiltration and multiple cavities at the left. *Fig. 6*—Bilateral first stage thoracoplasty.

respirations, 20 to 24. The sputum was positive; sedimentation rate, 29 vertical curve; blood picture within normal limits.

Examination of the chest revealed bilateral moderate apical dullness, cavernous breath sounds and crepitant rales (Fig. 7). Artificial pneumothorax was unsuccessful on the left side, consequently a two-stage thoracoplasty was performed. Nine months later, because of frequent hemoptyses and a positive sputum, an intrapleural pneumothorax was instituted on the right side. To bring about a more effective collapse a pneumonolysis was also performed (Fig. 8). The patient improved considerably, his sputum became negative and the sedimentation rate dropped to 16 diagonal line. This patient is at home and well.

## BILATERAL EXTRAPLEURAL PNEUMOTHORAX

*Case V.*—R. G., white female, aged 21 years, whose family history was negative. Patient had had several attacks of pleurisy.

She was admitted with a 3-year history of cough, expectoration of 2 oz. of sputum daily; occasional streaking and night sweats. The temperature ranged from 97 to 99° F.; pulse, 80 to 94; respirations from 20 to 26. Sputum, positive; sedimentation rate, 23 diagonal curve (Cutler method); blood picture within normal limits.

Examination of the chest revealed bilateral moderate dullness, cavernous breath sounds over the right and crepitant apex rales. Bronchial breath sounds and medium rales were audible over the left apex (Fig. 9). Artificial pneumothorax was unsuccessful on both sides. A right extrapleural pneumothorax was performed and four months later a left extrapleural pneumothorax was accomplished (Fig. 10). Marked improvement was noted in the patient's condition. The sputum became negative and the sedimentation rate at present is 18 diagonal line.

This patient is still under sanatorium care.

## EXTRAPLEURAL PNEUMOTHORAX AND THORACOPLASTY

*Case VI.*—J. C., a white male, aged 29 years. Family history and past history were negative.

The patient was admitted with a six months' history of cough, expec-

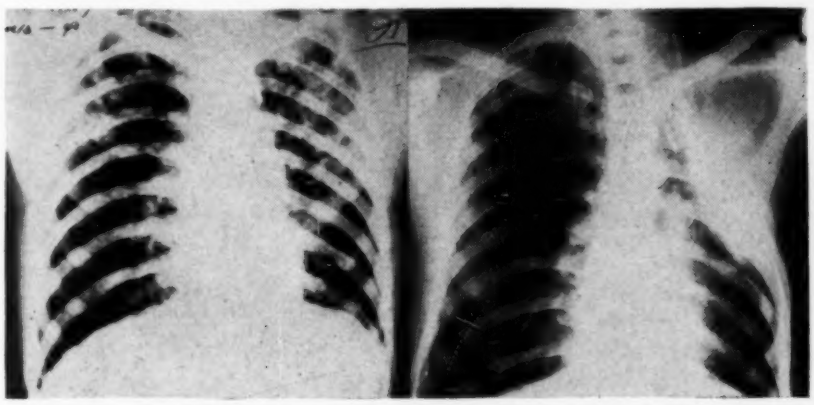

*Fig. 7* *Case IV* *Fig. 8*

*Fig. 7*—Bilateral infiltration and bronchopneumonic reaction of upper halves of both lung fields. *Fig. 8*—Left-sided second stage thoracoplasty. Partial pneumothorax of the right lung.

toration of 1 oz. of sputum daily; weakness and night sweats. The temperature ranged from 97 to 100° F.; pulse, 80 to 88; respirations, 20 to 22. The sputum was positive for tubercle bacilli; sedimentation rate, 28 vertical curve (Cutler method); blood picture within normal limits.

Examination of the chest revealed bilateral apical dullness, cavernous breath sounds and crepitant rales (Fig. 11). Artificial intrapleural pneumothorax was unsuccessful on the left side; consequently, a left-sided extrapleural pneumothorax was performed. The patient showed some improvement but the sputum remained positive because of disease in the right lung. Intrapleural pneumothorax was unsuccessful on the right side; and a two-stage thoracoplasty was performed (Fig. 12). Clinically the patient has shown considerable improvement. The patient still presents an occasional positive sputum and his sedimentation rate has dropped to 13 diagonal line. This patient is still in the sanatorium.

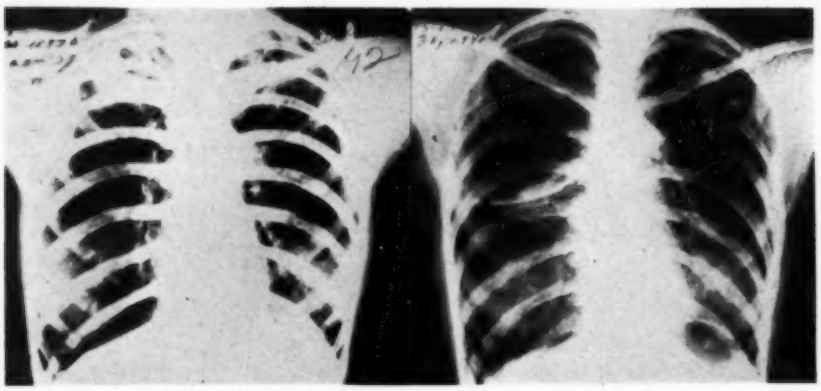

*Fig. 9* *Case V* *Fig. 10*

*Fig. 9*—Bilateral infiltration and cavitation involving upper third of both lung fields. (High contrast; insufficient detail.) *Fig. 10*—Bilateral extrapleural pneumothorax.

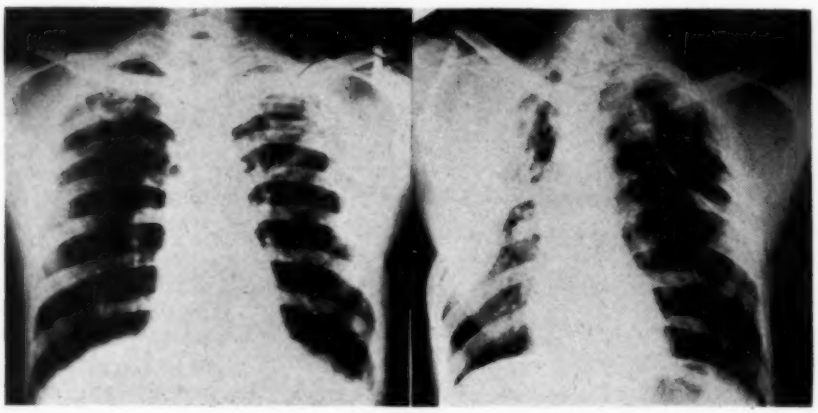

*Fig. 11* *Case VI* *Fig. 12*

*Fig. 11*—Infiltration and bronchopneumonic reaction of both upper lobes especially the left. Bilateral infraclavicular cavitation. *Fig. 12*—Left-sided extrapleural pneumothorax. Right-sided second stage thoracoplasty.

## EXTRAPLEURAL AND INTRAPLEURAL PNEUMOTHORAX

*Case VII.*—F. A., female, white, aged 27 years. Family history was negative. Past history of pneumonia.

The patient was admitted with an eighteen months' history of cough, slight expectoration, weakness and occasional night sweats. The temperature ranged from 98 to 99.6° F.; pulse, 78 to 90; respirations, 20 to 24. The sputum was positive for tubercle bacilli; sedimentation rate, 23 diagonal curve (Cutler method); blood picture within normal limits.

Examination of the chest revealed moderate dullness over the upper third of the right lung, bronchial breath sounds and medium rales. Slight dullness was obtained over the left apex (Fig. 13). Intrapleural pneumothorax was unsuccessful on the right side, consequently an extrapleural pneumothorax was performed.

In view of a persistent positive sputum and the presence of a cavity in the left lung in the region of the 2nd interspace an intrapleural pneumothorax was instituted. To bring about a more effective collapse a pneumonolysis operation was performed (Fig. 14). The patient improved both clinically and physically. The sputum became negative and the sedimentation rate dropped to 14 diagonal line. This patient is at home and well.

## SUMMARY

1) The only hope of arresting disease in the great majority of cases of bilateral pulmonary tuberculosis is by the use of effective collapse therapy.

2) The mortality in these cases if left alone is certainly very high, hence our justification for employing such extensive bilateral collapse therapy.

3) Careful selection of cases by both the phthisiotherapist and the thoracic surgeon is absolutely essential.

4) A satisfactory intrapleural pneumothorax is the treatment of choice. Pneumonolysis when indicated should always be performed.

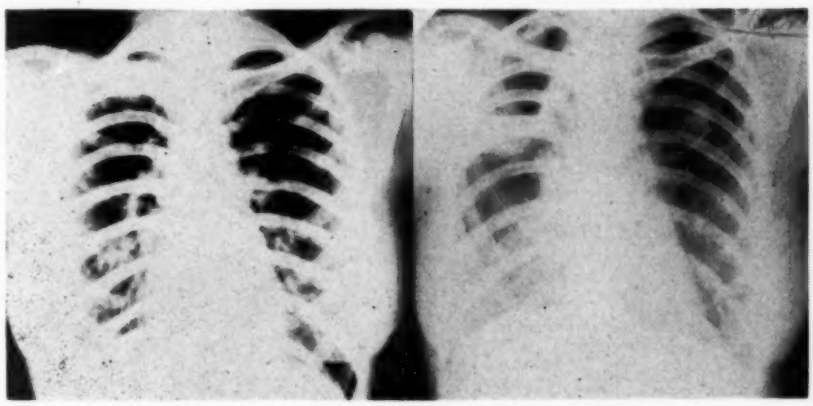

*Fig. 13* *Case VII* *Fig. 14*

*Fig. 13*—Infiltration and bronchopneumonic reaction of the right upper lobe. Slight infiltration in region of left second interspace at the periphery. *Fig. 14*—Right-sided extrapleural hydropneumothorax. Left-sided intrapleural pneumothorax.

5) When an intrapleural pneumothorax is unsuccessful a thoracoplasty or an extrapleural pneumothorax should be considered.

6) Basal lesions should be submitted to phrenic nerve surgery.

7) Oleothorax, as a means of collapse therapy, has not been very encouraging.

8) Bilateral collapse therapy is a safe, valuable and effective form of treatment and should be more universally employed.

9) Cases illustrating various combinations of bilateral collapse therapy have been presented.

## RESUMEN

1) La única esperanza de estacionar la enfermedad en la gran mayoría de los casos de tuberculosis pulmonar bilateral está en el empleo de la colapsoterapia eficaz.

2) La mortalidad en estos casos, cuando no se les hace nada, es ciertamente muy elevada, de aquí nuestra justificación para el empleo de colapsoterapia bilateral tan extensa.

3) La selección cuidadosa de los casos conjuntamente por el tisioterapéuta y el cirujano del tórax es absolutamente esencial.

4) El neumotórax intrapleural satisfactorio es el tratamiento de elección. Se debe ejecutar la neumonolisis siempre que esté indicada.

5) Cuando el neumotórax intrapleural es ineficaz debe considerarse la toracoplastia o el neumotórax extrapleural.

6) Lesiones basales deben someterse a la operación en el nervio frénico.

7) El oleotórax, como una forma de colapsoterapia, no ha dado resultados alentadores.

8) La colapsoterapia bilateral es una terapéutica segura, valiosa y eficaz y deber ser empleada más universalmente.

9) Se presentan casos ilustrativos de varias combinaciones de colapsoterapia bilateral.

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We are grateful to Dr. Horace Binney, visiting surgeon, and other members of the staff for assistance rendered in the preparation of this paper.

# Collapsed Lung—Postmortem Findings*

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J. WOODROW SAVACOOL, M.D.**
*Philadelphia, Pennsylvania*

Between 1935 and 1943, 30 cases of unilateral pneumothorax, 4 bilateral pneumothorax, 10 phrenic nerve paralysis, and 5 thoracoplasty were studied at autopsy by the authors.

The duration of collapse in these cases varied from 1 hour to 4 years. In the majority of the cases the collapse of the lungs was 50 per cent or more at necropsy. In a few the collapsed lungs were permitted to expand sometime before death. All the cases were observed clinically with frequent x-ray examinations of the chest. Just before the postmortem examination, almost all the cases were x-rayed again in order to compare the necropsy findings with the x-ray appearances. In 16 cases the pulmonary arterial system was injected with barium sulphate suspension and roentgenograms were made of them in order to obtain information regarding the gross pulmonary vascular changes. The technic was described in our previous study.[1]

## FINDINGS AND DISCUSSION

Inflammation of the pleura was a constant finding in all the cases. The normal lustre of the pleura was lost, the surface being covered with fibrinous exudate. Superficial tubercles were common, especially in the anterior surface of the lung; the posterior surface showed severe congestion, but definitely fewer caseous nodules. Numerous subpleural tubercles of varying sizes were easily seen. In the histological examinations tubercles were discovered not only in the subpleural zones, but in the pleura itself.

In 4 cases the caseous nodules situated near the anterior surface of the lung perforated through the pleura, apparently through the center of the tubercles. The pleura covering the nodules was thin and semi-transparent and free from adhesions. In following these perforations deeper into the pulmonary parenchyma, it was found that the tracts led into irregularly-shaped small, young cavities. These changes were most common anteriorly due perhaps to the ballooning-out effect of the anterior portion of the lung during

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**From the Department for Diseases of the Chest, Jefferson Hospital, Philadelphia, and White Haven Sanatorium, White Haven, Pennsylvania.

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respiration. Contributing to this mechanism is the attachment of the ribs to the rigid spinal column posteriorly and to the more flexible sternum anteriorly, which allows greater expansion of the lung in the front part of the chest. Also, when the patient lies on his back, the common practice, the above factors become more influential. In one case, a tear of the pleural adhesion between the mediastinum and the apical emphysematous blebs (Fig. 1) surrounding fibrotic tuberculous nodules was the cause of a small amount of serosanguinous effusion. The blood originated probably

*Fig. 1*—Three adhesions between emphysematous blebs and trachea. The arrow points to the perforation caused by rupture of an adhesion.

in the rupture of terminal bronchial arteries rather than the pulmonary vessels, since it is the bronchial artery which supplies capillaries to the pleura. Actual tear of the adhesions between the parietal and visceral layers of the pleura was found in 8 cases of chronic fibro-ulcerative tuberculosis with considerable pleural thickening; in each instance the tear was located along the axillary aspect of the lung.

Fibrosis of the pleura originated apparently in the parenchymal surface of the visceral pleura; projections of young fibrous tissue extending into the lung were noted. In the absence of acute exacerbation, the thickened pleura was unusually devoid of blood vessels, but in cases with acute relapse or spontaneous pneumothorax many blood vessels were seen within the thickened pleura. This suggested that occasional small recurrences of effusion in certain patients were due to the disturbances of the vessels.

The so-called selective collapse (Fig. 2a, b, c, d) has received much attention and various explanations have been advanced regarding its mechanism. The findings in our series suggested that this was essentially a phenomenon of atelectasis. In the diseased areas the bronchioles were greatly narrowed by exudative inflammatory changes; and when even a small amount of air was introduced into the pleural cavity, these bronchioles became completely occluded with absorption of the air peripheral to them. Selective collapse was seen more frequently in the exudative type of tuberculosis without fibrosis. In some of these cases cavitation developed in the center of the collapsed lung although the collapse was satisfactory. At times such a cavity becomes filled with caseous matter, and therefore difficult to be recognized clinically. Often pleural effusion and tuberculous empyema were complications.

In the collapsed lung there was a more or less uniform narrowing of the intrapulmonary bronchi and bronchioles, not necessarily from permanent anatomic changes, but from relaxation of the lungs in general. In a few cases of local constriction of the bronchi, tuberculosis was found to be the etiologic factor; and in such cases, caseous material had accumulated at the constricted portions of the bronchi. These patients had complained of noise in the chest, cough, difficulty in expectoration, paroxysmal dyspnea and stertorous breathing. The sputum had been persistently positive for tubercle bacilli.

In several cases of so-called giant, balloon or ball-valve cavities, the tuberculosis was acute with rapid break-down of the pulmonary tissue resulting in a multilocular cavity with bronchial stumps hanging free in the cavity. Air could be pumped into the cavity through the bronchus but it could not be expelled by pressing on the cavity wall. A similar mechanism presumably was present during life,

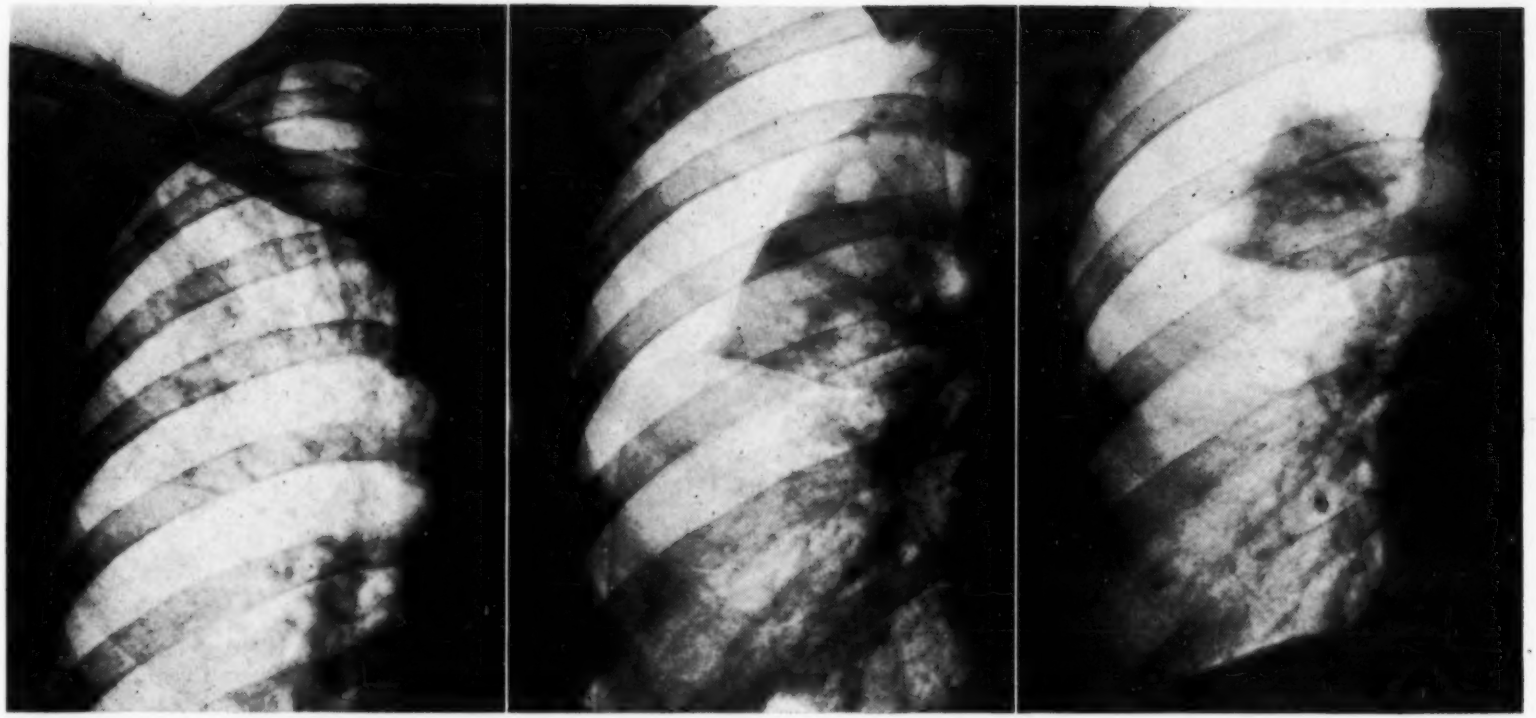

*Fig. 2a* *Fig. 2b* *Fig. 2c*

*Fig. 2a*—A female, aged 18. Exudative tuberculous lesions in right upper lobe with an apical cavity. *Fig. 2b*—Artificial pneumothorax with a selective collapse of the upper lobe. The cavity remains open. *Fig. 2c*—The adhesion was cut. Cavity disappeared with complete collapse of the upper lobe. *(Courtesy of Dr. Paul C. Swenson).*

permitting ingress of air during inspiration but preventing its escape during expiration. Fluid levels were common in these cavities. In another type, occurring in chronic smooth-walled cavities, there was no stump extending into the cavity but the bronchus ended in the fibrous wall of the cavity. The lumen was so narrow that it could not be seen easily, but by introducing a probe through the main bronchus, the opening could be found. In spite of such narrowing the proximal portion of the bronchus was fairly normal. Apparently during inspiration with the bronchial dilatation, air could enter the

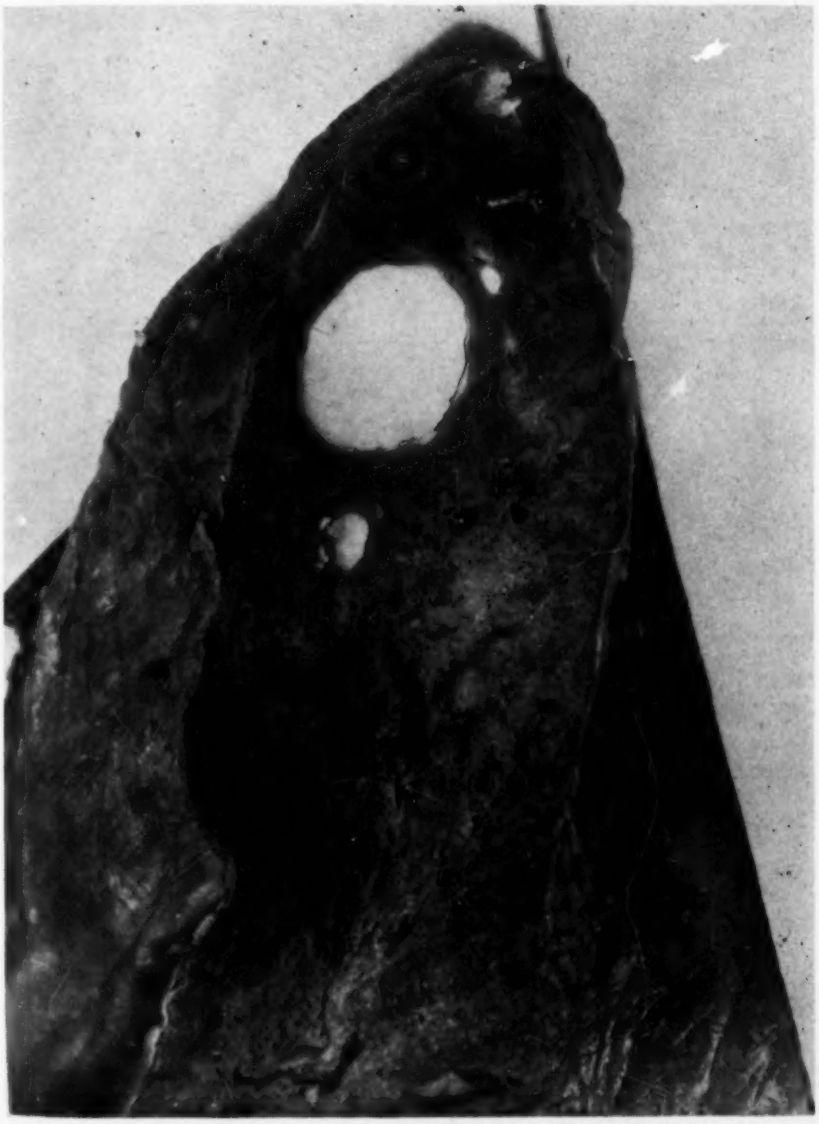

*Fig. 2d*—At autopsy the right upper lobe showed marked atelectasis. The cavity was not collapsed but filled with caseous material, thereby not appearing in the x-ray film.

cavity but during expiration with bronchial narrowing, air remained trapped in the cavity.

The pulmonary blood vessels (Fig. 3a, b, c, d) in the tuberculous areas became narrow and short. When the collapse had been present for more than six months, panarteritis was noted, particularly in and about the tuberculous areas. The intima showed thickening and this sometimes proceeded to thrombosis and occlusion of the vessel, with ultimate fibrosis. In the portions of the lungs which were free of tuberculosis, vascular changes were not noticeable, even when the lung had been collapsed for a long time. This suggested that vascular changes depended largely on the disease rather than on the collapse. The most extensive vascular sclerosis was noted about the fibrotic cavities. Such vascular changes with concomitant ischemia of the surrounding parenchyma seems to be a factor of the failure of such cavities to heal even under fairly satisfactory collapse. About the more recent cavities vascular changes were not pronounced and the surrounding parenchyma was well supplied with blood.

In 5 cases the main pulmonary arteries leading into the collapsed lung showed complete thrombosis. The symptoms following massive thrombosis of the pulmonary artery simulated those of spontaneous pneumothorax or of coronary artery occlusion. Such thrombosis occurs more often on the right side probably because of the anatomic relationship of the right artery to the arch of the aorta. On the right side the artery is crossed over by the arch of the aorta and at

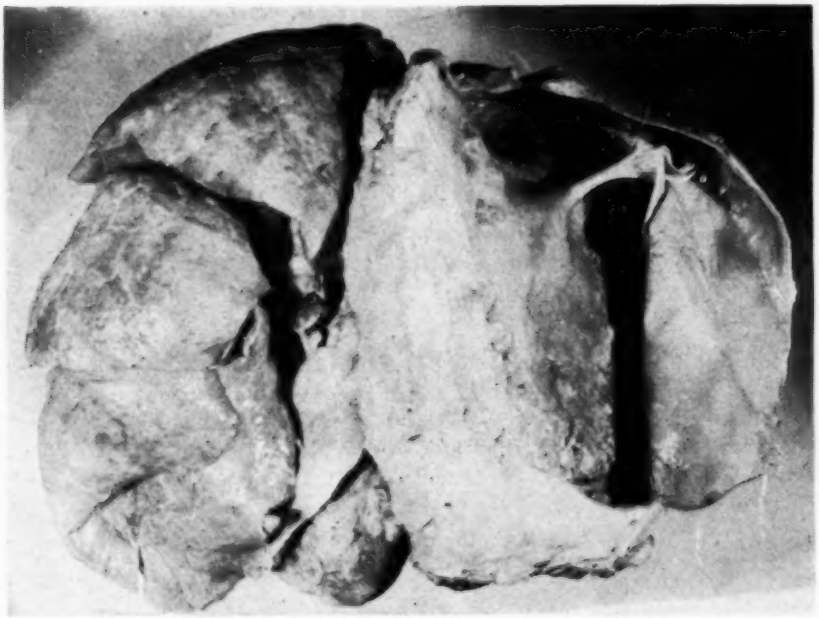

*Fig. 3a*—A collapse of the left lung with an adhesion.

the hilum it is "squeezed" between the vein and bronchus. On the left side the artery is relatively free.[2]

The branches of the intercostal vessels apparently supply the parietal portion of the adherent cavity walls. It has been observed clinically that when large and fibrotic cavities were made to collapse by separation from the chest wall by means of extra-pleural pneumolysis, empyema developed in a large number of cases, some months after the operation. In such cases bronchopleural fistulae were suspected. In a recent case studied postmortem, it was found that the entire peripheral portion of the cavity had sloughed, presumably on account of isolation of the cavity wall from its blood supply in the chest wall.

Tuberculous cavities heal apparently not by regeneration of lung tissue but by fibrosis. Our findings indicated that the cavities closed by concentric collapse and fibrosis which was brought about by purse-string-like constriction of a circular band of fibrous tissue

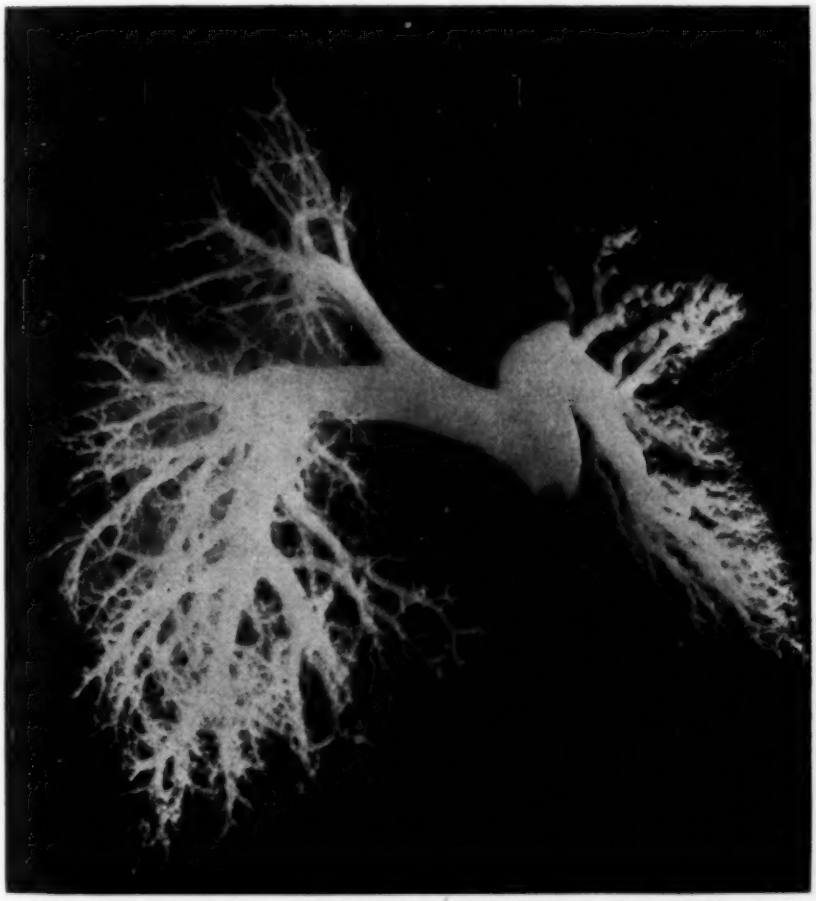

*Fig. 3b*—A pulmonary arteriogram showing marked narrowing and shortening of the pulmonary arteries in the collapsed lung.

formed outside the cavity wall. This process was favored by adequate blood supply and relaxation of the surrounding parenchyma. When the pulmonary structures surrounding the cavity were ischemic and fibrosed, the cavity instead of undergoing concentric collapse, flattened from side to side. When this occurred, permanent anatomic healing did not take place. In such cases searches were made for fibrous tissue crossing the flattened cavity thus suggesting a possible mode of healing but none was found. Relatively dry and somewhat flaky caseous material was often the content of such cavities.

In 10 cases of phrenic nerve paralysis there was severe fibrosis of the paralyzed diaphragm (Fig. 4) with extensive adhesion formation between the diaphragm and the adjoining lung. In the collapsed portion of the lower lobe there were vascular changes, thickening of the alveolar walls and narrowing of the bronchial lumens, but the anatomic changes were less noticeable than in pneumothorax. Definite bronchiectasis which might have been caused by diaphragmatic paralysis was not found. In none was there evidence of pulmonary abscess or gangrene from retention of secretions. Phrenic

*Fig. 3c*—A histologic section of the shortened pulmonary arteries showing marked sclerosis.

paralysis was found to bring about relatively greater rise of the central and posterior portions of the diaphragm than of the anterior portion. This suggests that the central and posterior cavities may be more easily collapsed by phrenic paralysis than the anterior cavities.

Thoracoplasty produced in general the same pulmonary changes as pneumothorax but they were more severe. In two cases in which death occurred a few days after operation, the exact cause of death was difficult to determine. There was no definite myocardial change, atelectasis, bronchial occlusion, pneumonia, or acute spread of the disease. It was assumed that great respiratory disturbance brought about by paradoxical respiration was the cause.

## COMMENT

These findings—namely, pleural reaction, bronchial narrowing, vascular sclerosis and parenchymal changes noted in these cases—were apparently the combined results of tuberculosis and collapse therapy. With a view to determine whether these changes may occur in non-tuberculous but collapsed pulmonary tissues, artificial pneumothorax was given in twelve non-tuberculous rabbits for a period of nine months. The only permanent anatomic change noted

*Fig. 3d*—A cross section of the adhesion, showing sclerosis of the vessels.

was non-inflammatory pleural thickening. There was nothing to suggest pulmonary parenchymal vascular and bronchial changes. This study will appear in another communication.

## SUMMARY

1) Pulmonary changes in collapse therapy were studied at autopsy in 49 cases.

2) Pleural inflammation with or without effusion was a constant finding and it was usually related to the tuberculous lesions in the underlying lung. Tuberculous empyema occurred from rupture of adhesions or of subpleural tubercles rather than from faulty operative technique.

3) Selective collapse was caused apparently by atelectasis resulting from occlusion of diseased bronchioles in the tuberculous areas.

4) Dilatation of the so-called giant or ball-valve cavities depended upon tuberculous changes of several types in the draining bronchi which permitted air to enter but prevented it from leaving the cavities.

5) Panarteritis, leading to thrombosis and sclerosis in the tuberculous areas was a constant finding and resulted in considerable loss of blood supply about the chronic fibrotic cavities. This appeared to

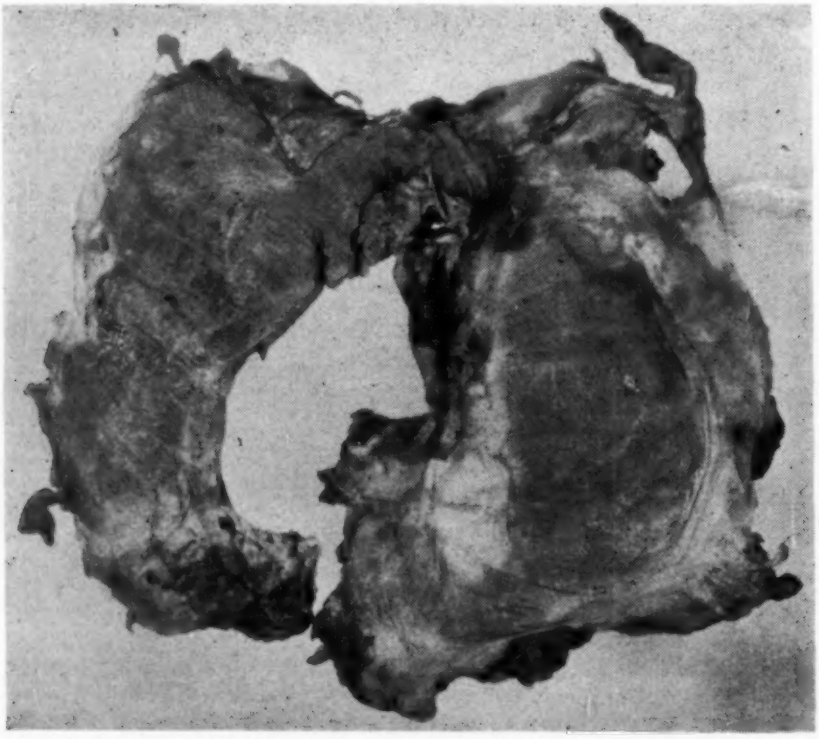

*Fig. 4*—The right half of the diaphragm was paralyzed. It shows marked thinning and fibrosis of the paralyzed half of the diaphragm.

be responsible for failure of such cavities to undergo healing even under satisfactory collapse.

6) Vascular changes around recent cavities were minimal, the blood supply being therefore practically unimpaired. This appeared to contribute to the healing of such cavities which occurred by concentric collapse and fibrosis.

7) Phrenic nerve paralysis caused extensive fibrosis of the paralyzed portion of the diaphragm and brought about relatively greater rise of the central and posterior portions of the diaphragm than of the anterior portion. Consequently the posterior and central cavities were more influenced by phrenic paralysis than the anterior ones.

8) It is suggested that collapse therapy in chronic pulmonary tuberculosis should be carried out before vascular and fibrotic changes are too great to permit concentric cavity closure and anatomic healing. Serious complications almost never occurred in early tuberculosis in which there was satisfactory collapse of the lesions.

## COMENTARIO

Estos hallazgos—a saber, la reacción pleural, el estrechamiento bronquial, la esclerosis vascular y las alteraciones parenquimatosas observadas en estos casos—aparentemente fueron el resultado de la combinación de la tuberculosis y la colapsoterapia. A fin de determinar si estas alteraciones podrían sobrevenir en tejidos pulmonares colapsados pero no tuberculosos, se aplicó el neumotórax artificial a doce conejos no tuberculosos durante un período de nueve meses. La única alteración anatómica permanente que se observó fue un engrosamiento pleural no inflamatorio. No hubo ninguna indicación de alteraciones pulmonares parenquimatosas, vasculares o bronquiales. Este estudio aparecerá en otro informe.

## RESUMEN

1) Se estudiaron las alteraciones pulmonares por la colapsoterapia reveladas en autopsias de 49 casos.

2) Inflamación pleural con derrame, o sin él, fue un hallazgo constante que se observó generalmente en relación con las lesiones tuberculosas en el pulmón subyacente. Cuando sobrevino empiema tuberculoso fue debido más bien a rotura de adherencias o de tubérculos subpleurales que a errores técnicos.

3) Colapso selectivo fue aparentemente causado por atelectasia debida a la oclusión de bronquiolos afectados en las zonas tuberculosas.

4) Dilatación de las cavernas llamadas gigantes o de tipo de válvula de retención fue debida a lesiones tuberculosas de varios tipos en los bronquios de desagüe que permitían la entrada de aire a estas cavernas pero impedían su salida.

5) Panarteritis, que causó trombosis y esclerosis en las zonas tuberculosas, fue un hallazgo constante que resultó en una disminución marcada de la circulación sanguínea alrededor de las cavernas fibrosas crónicas. Esta pareció ser la razón por la cual no se pudo obtener la curación anatómica de estas cavernas aún existiendo colapso satisfactorio.

6) No se observaron alteraciones vasculares marcadas alrededor de cavernas de origen reciente y, por consiguiente, la circulación sanguínea se halló prácticamente intacta. Esto apareció contribuir a la curación de estas cavernas y se produjo por medio de colapso concéntrico y fibrosis.

7) Parálisis del nervio frénico causó fibrosis marcada de la parte paralizada del diafragma y produjo relativamente mayor elevación de las partes central y posterior del diafragma que de la parte anterior. Por consiguiente las cavernas posteriores y centrales fueron afectadas más por la parálisis del frénico que las anteriores.

8) Se sugiere que la colapsoterapia en la tuberculosis pulmonar crónica debe aplicarse antes de que las alteraciones vasculares y fibrosas sean tan extensas que impidan el cierre concéntrico de las cavernas y la curación anatómica. Casi nunca sobrevinieron complicaciones graves en tuberculosis incipiente con colapso satisfactorio de las lesiones.

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# Some Clinical Problems in Bronchiectasis*

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Physicians who have made thoracic disease their principal interest fortunately have been privileged to witness, in recent years, developments which should be recorded as great milestones in medical history. Among these developments one can mention surgical treatment of pulmonary tuberculosis and the multiplication of facilities for such treatment to such a degree that in many communities all patients with tuberculosis may have the benefit of collapse therapy if they need it. Great forces have been put into motion which eventually may result in control of tuberculosis to a degree similar to that already attained for many other infectious diseases. Surgical conquest of thoracic disease also has been extended to include malignant disease within the thorax in a steadily increasing number of cases.

The newer knowledge of bronchiectasis deserves even greater emphasis than some of the preceding conditions, because a smaller proportion of the medical profession has come to know what great improvements have been made in the prevention, diagnosis and treatment of this extremely important disease. It is the definite responsibility of physicians who specialize in diseases of the thorax to disseminate this information among their fellow physicians whose primary interest is not in the field of thoracic disease. Physicians in general practice have the responsibility of recommending the necessary procedures which eventually will establish the diagnosis of bronchiectasis and which will in turn lead to proper treatment. We must emphasize that in a reasonable number of cases of bronchiectasis the disease is curable by radical means and that the curability of the disease is dependent upon early recognition; that is, while it is still localized and while the patient is sufficiently young to undergo surgical treatment with minimal risk. Physicians in general practice are eager to learn what is known about the prevention of permanent pulmonary damage that follows acute pulmonary infection. Patients and physicians alike are learning that chronic productive cough is a symptom which demands explanation in every instance and that it often cannot be properly ascribed to "chronic bronchitis" or "sinusitis" until the actual anatomy of the

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**Division of Medicine, Mayo Clinic.

tracheobronchial tree has been studied by bronchoscopic and bronchographic means.

## PROGNOSIS

The prognosis of bronchiectasis is difficult to state, not only because of the wide variation in individual cases but because of limited experience with cases of truly proved bronchiectasis for a long time. The use of iodized oil as a diagnostic agent was introduced in 1922, but it has not been widely employed except during the past ten or fifteen years. Studies which have been made to determine the actual mortality rate of bronchiectasis have not usually included cases of mild forms of the disease, in which the patients might not seek medical care. Such cases would greatly dilute the statistics if included. However, the literature on the subject is almost unanimous in agreeing that bronchiectasis has a high mortality rate, in addition to producing serious disability. There is no way of measuring the reduction in earning capacity and the blighting of human happiness produced by this debilitating and loathesome disease. If we made an effort to average the figures given by various students of the disease[1] we might conclude that less than 10 per cent of patients with severe bronchiectasis obtain a satisfactory result from any form of medical treatment and that the mortality rate is somewhere between 30 and 50 per cent within ten or fifteen years after the diagnosis is made. We may state, furthermore, that the majority of patients with severe bronchiectasis are destined to die of the disease or of a complication such as pulmonary abscess or pneumonia. When severe bronchiectasis appears in childhood it is improbable that the child will live beyond the age of forty years.

## ETIOLOGY

It is extremely important that every patient who has an acute pulmonary infection, such as pneumonia, should be followed roentgenographically until the lesion has completely disappeared. This is essential, particularly in the case of children and infants, and should be stressed regardless of the fact that symptoms may have ceased entirely. If infiltration has persisted for several weeks or longer after such an infection, the possibility of incipient bronchiectasis must be considered. This is especially true if the lesion has any appearance of atelectasis or if physical findings suggest incomplete aeration. The time may come when physicians will feel dependent upon the roentgenogram for clinical guidance in cases of acute pulmonary infections as they do now in the management of tuberculosis. No modern physician would accept clinical evidence of healing of a tuberculous lesion if this evidence conflicted with

roentgenographic evidence, or would he feel secure in giving advice to patients without continual reference to roentgenographic changes. If a similar attitude is taken toward acute infections which resemble pneumonia and influenza, it should be possible to prevent many instances of acquired bronchiectasis.

Anspach[2] and Holinger[3] have shown that postpneumonic bronchial obstruction and atelectasis may be forerunners of true bronchiectasis, especially in infants and children. It is possible to perform bronchoscopy on these patients and establish drainage within a few weeks and to avoid the tragedy of progressing bronchiectasis. The necessity for bronchoscopy is not always apparent on physical examination or upon the history alone, but roentgenologic studies will frequently reveal the need for such investigation and treatment.

It should be emphasized that infants under the age of one year have a poorly developed cough reflex and that the caliber of their bronchi is so small that obstruction is more likely to develop than in later life. A cicatricial narrowing of a bronchus may occur secondary to acute or chronic tracheobronchial inflammation and, when established, impedes endobronchial drainage distal to the site of obstruction. This permits the establishment of chronic infection in this portion of the bronchial tree, which may destroy essential irreplaceable structures in the walls of bronchi. The result is not unlike that produced by obstruction by a foreign body which has long been retained or that produced by tumor or extrinsic pressure upon a bronchus.

Not only may pneumonia be the forerunner of bronchiectasis but patients with bronchiectasis who have intermittent episodes of bronchial obstruction have symptoms which frequently are clinically similar to those produced by recurrent attacks of pneumonia. Any patient who has recurrent illness with respiratory symptoms associated with chills and fever may be suspected of having intermittent bronchial obstruction. Frequently, but not always, such patients will have continuous chronic productive cough, and these acute episodes may be associated with temporary suppression of expectoration, but when the fever declines and drainage is re-established, the sputum becomes more abundant and purulent. Patients who have such symptoms require bronchoscopic examination regardless of the fact that there may be no abnormal physical findings and no abnormalities may be noted upon roentgenographic examinations of the thorax. We have found bronchoscopy to be of increasing value in cases of acute recurrent pulmonary infection and in postpneumonic sequelae as well as in more chronic affections of the tracheobronchial tree.

There is no longer any doubt that there are true congenital

varieties of bronchiectasis due to development anomalies. Olsen[4] recently has reviewed the syndrome known as Kartagener's triad, which consists of an association of transposition of the viscera, bronchiectasis and sinusitis. He reviewed a series of eighty-five cases of congenital dextrocardia and found that in fourteen, or 16.5 per cent, of these cases, there also was definite evidence of bronchiectasis. In these eighty-five cases, the incidence of bronchiectasis was thirty or thirty-five times as high as it is in the general run of patients who register at the Mayo Clinic. In addition to these fourteen cases of bronchiectasis associated with dextrocardia, thirty-one similar cases previously were reported by various authors. Many physicians also are familiar with another congenital disease, so-called "fibrocystic disease of the pancreas," which is almost invariably associated with severe, extensive bronchiectasis. This condition has recently been reviewed by Kennedy,[5] and the congenital, developmental nature of bronchiectasis in these cases appears to be probable. We also have seen similar bronchiectasis appear in identical twins and the evidence of a congenital type of disease again was very suggestive. Many other patients with bronchiectasis describe lifelong cough and expectoration, which sometimes lead to a suspicion that the disease is congenital, but it usually is not possible to exclude the development of acquired bronchiectasis in early childhood.

## BRONCHIECTASIS OF THE UPPER LOBE

Bronchiectasis that is limited to an upper lobe is of unusual interest because of the entirely different clinical picture which is sometimes seen and because of the frequency with which the disease might be confused with tuberculosis. Bronchiectasis of the upper lobe without involvement of a lower lobe is an unusual diagnosis to make, but we have suspected that it may be a more frequent condition than generally is recognized, since it may occur in the absence of chronic expectoration and in the absence of any abnormal findings in an ordinary roentgenogram. The patients with bronchiectasis of an upper lobe may seek medical attention because of recurrent pulmonary hemorrhage which could not be explained by ordinary methods of investigation. They may have an irritative cough, but sometimes deny the presence of chronic cough and expectoration. It seems possible that the excellent drainage of the upper lobe afforded by gravity might explain the failure of secretion to accumulate, to become infected, and to produce the characteristic symptoms of septic bronchiectasis. The diagnosis of bronchiectasis of the upper lobe usually can be made only by bronchographic examination with iodized oil. Bronchiectasis of the upper lobe with minimal cough and expectoration again emphasizes the importance

of postural drainage as a method of treatment of lesions of the lower lobe that are not suitable for surgical treatment.

## HEALING OF BRONCHIECTASIS

Before subjecting a patient to lobectomy or pneumonectomy, which carries a reasonable but definite mortality rate, one must first decide whether or not nonsurgical treatment offers any reasonable possibility of permanent cure.

If one reviews the pathologic picture of true bronchiectasis, one sees how difficult it is for Nature to repair such damage. There has been extensive destruction of irreplaceable structures in the walls of important bronchi with resultant functional impairment of the air channels and accumulation and putrefaction of secretion, which lead to further destruction and greater functional impairment. It is true that in rare instances the element of infection may come under temporary control sufficiently to simulate a cure. This occurs in cases of so-called dormant bronchiectasis. It is difficult to believe that the mechanical defects have been repaired and it is impossible to reassure the patients that they are safe from recurrence of their symptoms. Many of them return later because of a recurrence of symptoms. Follow-up studies for a sufficiently long period have been made in only a few cases in which the diagnosis originally was proved by bronchographic studies by means of iodized oil. We have known patients with bronchiectasis to have been relieved of symptoms for as long as fifteen years, only to suffer recurrence following a superimposed acute pulmonary infection.

A more common cause for apparent cure of bronchiectasis is a mistake in diagnosis and a failure to recognize that infection of the paranasal sinuses may lead to symptoms which are indistinguishable from those of true bronchiectasis. In such instances it appears that purulent material which is draining from these sinuses is aspirated into the tracheobronchial tree, especially during sleep, and produces aspirational bronchitis and leads to voluminous morning expectoration quite similar to that experienced by patients with bronchiectasis. Under these conditions relief of the sinusitis also will relieve the pulmonary symptoms. The diagnostic distinction here is dependent upon bronchographic studies with iodized oil. Although it is entirely possible that sinus infection may eventually lead to true bronchiectasis, we believe it to be even more frequently true that when the two conditions are associated the sinus infection is secondary to the bronchiectasis.

If iodized oil is introduced into the bronchi of a lobe that is atelectatic, an illusion of bronchiectasis may result owing to a foreshortening and apparent widening of the larger bronchi. This appearance may resemble that of cylindrical bronchiectasis. When

the lobe re-expands and the bronchi stretch to their normal length, the caliber is correspondingly reduced and what simulated bronchiectasis again becomes normal; however, this does not represent true healing of bronchiectasis.

## DIAGNOSIS

We cannot emphasize too strongly the extreme importance of distinguishing basal pulmonary lesions which are due to carcinoma but which may closely simulate bronchiectasis. Even though such lesions have all of the roentgenographic characteristics of an inflammatory lesion, the possibility of carcinoma must be given serious consideration if the infiltration is unilateral. If such a unilateral basal lesion is of recent origin, it is more likely to be carcinomatous than if symptoms or previous studies demonstrated its existence at a remote previous time. If symptoms are few or are mild or absent, there is still a possibility that the lesion is a carcinoma. If the age of the pathologic process cannot be determined, but if symptoms are progressive, the probability of carcinoma is further increased. If the symptoms are insidious in their development and are not associated with any obvious acute respiratory infection, the possibility of carcinoma is also enhanced. Although bronchiectasis of benign nature may appear insidiously and progress, true bronchiectasis usually can be traced to some acute respiratory infection, such as pneumonia or pulmonary abscess, with incomplete resolution of symptoms.

We must emphasize that the roentgenologist is frequently unable to distinguish an inflammatory lesion from a carcinomatous lesion. We feel sure that this is due to the fact that the visible lesion itself is frequently bronchiectasis produced by bronchial obstruction occasioned by the presence of a tumor. In cases in which symptoms of bronchial obstruction have been present for a relatively long time, for example, a few years, it is not infrequent to discover that the patient has a carcinoma of a low grade of malignancy. It seems wise to suggest that the roentgenologist should mention in his report the possibility of carcinoma in every instance of unilateral basal infiltration even though it has the appearance of an ordinary inflammatory lesion or common bronchiectasis. Diagnostic bronchoscopy will be required in all such instances to exclude the possibility of an obstructing bronchial lesion.

Although basal tuberculosis does occur and can simulate bronchiectasis, and although it is extremely important that sputum be examined in every instance for mycobacterium tuberculosis, carcinoma still appears to be a more likely diagnosis than tuberculosis in cases in which a lower lobe is the sole site of an apparently inflammatory process and when the sputum is negative. We have

known patients with carcinoma to lose many valuable months undergoing treatment for suspected basal tuberculosis in sanatoriums when early bronchoscopy would have established the diagnosis and permitted radical surgical removal of the lesion.

In our experience, roentgenographic studies with iodized oil are of limited value in excluding obstructing lesions of a bronchus. Although the site of obstruction may be demonstrated bronchographically, it is frequently impossible to recognize the obstruction, and incomplete filling of bronchi may simulate organic obstruction in cases in which such obstruction is not present. When an obstructing lesion is suspected, bronchoscopy should always precede bronchography.

We recommend bronchoscopy in most cases of bronchiectasis with evidence of unilateral infiltration, in cases of recurrent attacks of respiratory infection with chills and fever, and in many other cases in which the possibility of a long retained foreign body or other type of obstruction may exist.

## SUMMARY

Bronchiectasis is a serious disease, frequently a fatal one, but is not accorded the attention that it deserves. It is frequently preventable, is occasionally curable, and may be difficult to diagnose accurately. Symptoms of bronchiectasis may be confused with those of chronic infection of the upper part of the respiratory tract. Bronchiectasis that is confined to an upper lobe may be very atypical, clinically and roentgenologically. Bronchiectasis may be caused by postpneumonic bronchial obstruction, obstructing bronchial tumors, long retained foreign bodies and developmental defects. True bronchiectasis appears to be incurable by any medical treatment and requires surgical extirpation. Selection of patients for operation requires close cooperation between the internist, the roentgenologist, the bronchoscopist and the surgeon. Bronchoscopy is frequently required and bronchographic studies with iodized oil are usually necessary to properly evaluate the situation. Many instances of "postpneumonic" bronchiectasis might be prevented if all patients with acute pulmonary infections were followed roentgenographically after clinical recovery. It is suggested that clinical judgment should be guided by roentgenography in management of acute pulmonary disease as it now is in the management of tuberculosis.

## RESUMEN

La bronquiectasia es una enfermedad grave y frecuentemente es fatal, pero no se le presta la atención que merece. Con frecuencia es prevenible, de vez en cuando es curable, y a veces es difícil de diagnosticar con exactitud. Los síntomas de la bronquiectasia pue-

den ser confundidos con los de infección crónica de la parte superior del aparato respiratorio. Bronquiectasia limitada a un lóbulo superior puede ser muy atípica desde los puntos de vista clínico y roentgenográfico. La bronquiectasia puede ser causada por obstrucción bronquial postneumónica, tumores bronquiales obstructores, cuerpos extraños retenidos por largo tiempo y defectos en el desarrollo. La bronquiectasia verdadera no parece ser curable por ningún tratamiento médico y requiere extirpación quirúrgica. La selección de pacientes para la operación requiere una cooperación íntima entre el internista, el roentgenólogo, el broncoscopista y el cirujano. Con frecuencia se requiere el examen broncoscópico y generalmente se necesita hacer estudios broncográficos con aceite yodado para avaluar la situación correctamente. Se podría prevenir muchos casos de bronquiectasia "postneumónica" si a todos los pacientes con infecciones pulmonares agudas se les estudiara por medio de radiografías después de la reposición clínica. Se sugiere que el juicio clínico debe guiarse por la roentgenografía en el tratamiento de las neumopatías agudas, así como lo es al presente en el tratamiento de la tuberculosis.

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# The Role of the Bronchial Tree in the Pathogenesis of Pulmonary Tuberculosis*

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The efficient manner in which the normal bronchial tree cleanses itself of foreign material under ordinary conditions has long been known, but due consideration has not been given it in pulmonary tuberculosis. It will be my endeavor, therefore, to emphasize the normal function of the bronchial tree in the prevention of reinfection tuberculosis. It will be my attempt, also, to show how unfavorable conditions affecting this normal mechanism may be responsible for the retention of tuberculous exudates and how inadequate drainage results in the development of disease with spread to the pulmonary bed distal to the blockage.

Several investigators have observed the presence of a very complex peristaltic-like action in the tracheobronchial tree. Macklin[1,2] has described "an elongation of the bronchial trunk and its branches during inspiration and a corresponding shortening of these structures during expiration; a dilatation of the bronchi during inspiration, narrowing during expiration; a downward motion of the lower end of the trachea and main bronchi and lung root, in passing from expiration to inspiration, and an upward motion in passing from inspiration to expiration." Macklin emphasized, also, the "restricted movement of the bronchial tree in the area of the human lung which lies between the root zone and the fixed wall behind and above the root region." This is the area in which tuberculosis so commonly develops and progresses.

Hudson and Jarré[3] demonstarted in iodized oil studies the lengthening and shortening of the bronchial tree during respiration. At the end of inspiration the iodized oil was clearly seen to fill the smaller bronchioles, whereas during the expiratory phase the bronchi and bronchioles shorten and contract and the iodized oil was no longer seen in the smaller bronchioles. These authors also demonstrated an absence of this "peristaltoid" movement in ectatic bronchi.

William Snow Miller[4] found that the bronchial musculature is arranged in a "network of geodesic bands which prevent any tangential motion and, in this way, provides for the greatest amount of extension and contraction of the bronchi. The muscle bands form a sphincter about the openings of the alveoli into the bron-

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chioli respiratorii and ductuli alveolares and also about the openings of the atria." He concluded that the action of the bronchial muscle in expiration is active and not passive and he remarked that its action in the regulation of the tension of the air within the atria and air sacs deserved more attention.

According to the above authors, the evacuation mechanism of the bronchial tree consists, therefore, in addition to the well known ciliary action, and the coughing mechanism, of a "peristaltoid" movement of the bronchial wall produced by the bronchial musculature and assisted by the elastic tissue. Any crippling of this mechanism would lead to a retention of infectious exudates within the bronchial tree.

Let us consider the possible sequence of events which take place following the inhalation of tubercle bacilli from an exogenous source into the lungs of an individual already primarily infected. The bacilli, under these conditions, are not treated as bacilli of first infection which, like dust particles, are phagocytized and carried by monocytes to the lymphoid tissue situated in the interstices of the lung. This is believed to be the chief protective mechanism in the prevention of the extension of primary tuberculosis to other parts of the lung. Bacilli of reinfection, on the other hand, together with their exudates formed as a result of allergy, remain within the bronchioles or other open air spaces and have every opportunity to be evacuated from the lungs. Under ordinary conditions an individual does not inhale into his lungs at any one time an overwhelming dose of tubercle bacilli. The exudate formed as an allergic response to them is therefore not usually large enough to block the bronchioles. Since it takes days for tubercle formation to occur it is believed that the normal bronchial evacuation mechanism may be truly as effective in protecting the individual from reinfection of exogenous origin as the lymphatic system is effective in preventing reinfection from primary foci situated in the interstices of the lung.

It is believed, therefore, that the requisites for the development of progressive pulmonary tuberculosis are: (1) reinfection with the tubercle bacilli; (2) the formation of tuberculous exudates within the lung and bronchial tree as a response to allergy, and (3) the mechanical failure of the bronchial tree to evacuate the infectious exudates. This mechanical failure of the bronchial tree to cleanse itself is due basically to the blockage of the bronchi or bronchioles by the tenacious exudate. Certain factors which might have a definite influence on the retention of infectious exudates within the bronchial tree and which might well play a leading role in the pathogenesis and localization of pulmonary tuberculosis will be discussed as follows:

1) Physical obstruction and developmental abnormalities.

2) The degree of allergic response of an individual to reinfection.

3) A difference in degree of functional ability of the bronchial tree in the apex and in the base of the lung, and its influence on the localization of pulmonary tuberculosis.

4) Alteration of the function of the bronchial tree following collapse therapy and its effect on drainage.

5) The effects of pathological changes within the parenchyma or within the interstitial portion of the lung on the drainage mechanism.

## PHYSICAL OBSTRUCTION AND DEVELOPMENTAL ABNORMALITIES

Physical obstruction to a bronchus or bronchiole by endobronchial cancer or endobronchial tuberculosis produces sequelae which are clearly understood and yet the same mechanical principle of obstruction has not been applied with equal significance to tuberculous exudates to explain the spread of pulmonary tuberculosis. Abscess formation is a common complication of endobronchial cancer when the malignant lesion is large enough to produce stenosis. Where bronchial tuberculosis exists in the presence of minimal tuberculosis the chances are that the lesion within the lung will not spread as long as adequate drainage occurs. If bronchial tuberculosis causes sufficient stenosis, however, a marked pneumonia will develop rapidly distal to the stenosis. Breaking down of the lung tissue with cavity formation may take place with unusual rapidity as a result of lack of drainage, and clinically the patient may be very ill. This demonstrates most clearly the mechanical side of the spread of tuberculosis, and further shows that acute rapidly spreading tuberculosis is not, necessarily, as previously believed, due to a lack of resistance or immunity of the individual to the disease.

It is reasonable to believe, also, that other less obvious physical or developmental changes within the bronchial tree may be responsible for a retention of tuberculous exudates with the development and spread of tuberculosis. A developmental widening or narrowing of a bronchus or bronchiole, or a kinked or markedly curved bronchus or bronchiole may function less efficiently with limitation of the "peristaltoid" movement and contraction of the bronchus in question. These abnormalities might well favor the implantation of bacilli and the retention of their exudates.

## THE DEGREE OF ALLERGIC RESPONSE OF THE INDIVIDUAL TO REINFECTION

The amount of exudate thrown out within a portion of the normal bronchial tree is dependent, usually, upon the degree of allergic response to the bacilli of reinfection. If this allergic response is

great, as in the Negro, blockage of the bronchioles occurs quickly and rapid spread to the pulmonary bed distally takes place in a manner similar to that which occurs where physical stenosis by endobronchial lesions exists. In addition, the tuberculous pneumonic process, which is so commonly seen in the Negro, greatly inhibits the elasticity of the lung and helps to prevent the evacuation mechanism from functioning. Progressive disease results. The degree of allergic response of an individual to reinfection is very important, therefore, in determining whether the bronchial tree will be functionally able or not to set up adequate drainage. Upon this will depend the course of the initial lesion, whether it will clear or whether it will progress. The greater allergic response of the Negro to reinfection may be due to several factors which are not well understood.

## A DIFFERENCE IN DEGREE OF FUNCTIONAL ABILITY OF THE BRONCHIAL TREE IN THE APEX AND IN THE BASE OF THE LUNG, AND ITS INFLUENCE ON THE LOCALIZATION OF PULMONARY TUBERCULOSIS

This has been discussed in detail by the author in previous publications. Our conclusions[5,6,7,8] on the pathogenesis and usual localization of pulmonary tuberculosis in the upper part of the lung have been based on a study of over 200 patients treated in the Waverly Hills Sanatorium since 1935 by simply raising the foot of their beds. The patients selected have been those with disease chiefly located in the upper part of the lungs, and showing inadequate drainage from the bronchial tree in this area. These patients with very difficult expectoration, irritable cough, shortness of breath, and inability to relax served to illustrate most clearly the beneficial effects of this position in the treatment of their disease. In 85 per cent of our patients with difficult expectoration treated in this manner ease of expectoration followed shortly the elevation of the foot of the bed. The other symptoms mentioned improved or disappeared in like percentage following ease of expectoration.

Increase in intra-abdominal pressure which follows the elevation of the foot of the bed results in a definite rise of the diaphragm. This rise in the diaphragm relieves the restricted mobility of the apical portion of the lungs and indeed allows for a more evenly distributed expansion and relaxation of the lungs during respiration. The upper part of the lung now functions more normally with greater lengthening and shortening movements of the bronchial tree. This results in a natural elimination of the accumulated infectious exudates in the otherwise less mobile apex. Sputum previously difficult to raise now literally wells up into the throat as more active movements of the bronchial tree in the apex are re-

sumed. It was concluded in previous publications by the author[5,6,7,8] that retention of the infectious exudate in the bronchi and bronchioles in the upper third of the lung, due to comparative lack of mobility of the lung in this region, is responsible, not only for the prevalence of tuberculosis in this region, but for its progression and chronicity. The base of the lung is usually not initially involved in a tuberculous process because of the very active mobility of the lung in this region. The base of the lung, however, may be very effective under adverse conditions in expelling exudates following serious bronchogenic spreads. We have all seen such spreads to the base from an active lesion in the upper part of the lung. Such basal lesions may often clear while the original upper lobe lesions remain unchanged.

## ALTERATION OF THE FUNCTION OF THE BRONCHIAL TREE FOLLOWING COLLAPSE THERAPY AND ITS EFFECT ON DRAINAGE

In a previous publication by the author[7] it was stated that artificial pneumothorax is successful when the collapse is good, not because the lung is put at rest but because compression of the lung forces out infectious exudates which the movements of the lungs and bronchial tree during respiration were unable to evacuate. By ridding the bronchial tree of these infectious exudates and by collapsing uninvolved lobules, a retrograde spread of disease into these lobules is prevented. Following initial pneumothorax refills the sputum may temporarily increase in amount, due to the compression of the lung. If the collapse is successful, there is no further spread of the disease. The bronchi remain patent and after a time exudate is no longer produced. If collapse is partial and open cavity or active disease is present, exudates will continue to develop and, due to loss of elasticity of the lung following partial pneumothorax, the exudates may be very difficult to raise. The impaired coughing reflex and the loss of the lengthening and shortening movements of the bronchial tree during respiration favor the stagnation of exudate within the lungs and bronchial tree of the partially collapsed lung.

In our original publication in 1938[5] in which we discussed the elevation of the foot of the bed in the treatment of pulmonary tuberculosis, emphasis was placed on the rise of the diaphragm and its mobility in explaining the cleansing mechanism of the bronchial tree, particularly in the apex of the lung. It is believed that patients who have had a previous phrenic paralysis are not able to evacuate these exudates as well as those who have not lost the function of the diaphragm. This is particularly true of patients who still have open cavity or other active disease following phrenic paralysis. Since our publication numerous writers have emphasized the im-

portance of the normally functioning diaphragm in thoracoplasty cases where the coughing mechanism is so necessary in cleansing the bronchi of accumulated exudate. It has been our view for several years that a normally functioning diaphragm is better in most individuals than a paralyzed one. Fundamentally it is believed that one of the important functions of the diaphragm is to aid in cleansing the bronchial tree of foreign matter which might accumulate within it. If the normally functioning diaphragm is essential in cleansing stagnated exudate from the bronchial tree in certain patients with thoracoplasty, then it must follow that it is necessary, but perhaps to a lesser degree, to the cleansing of the bronchial tree in normal individuals and in tuberculous individuals whose lungs have not been collapsed. It must be stated, however, that where phrenic paralysis results in the closure of cavity good results will be obtained, since further exudate will not be formed. If cases are not properly picked for phrenic paralysis, thick-walled cavities may remain open and these patients will be left at a disadvantage because of a lessened cleansing power of the bronchial tree. Most of the early bilateral phrenic paralysis cases did poorly, and, no doubt, for the reasons given above. The difficulty of the bronchial tree to cleanse itself of tuberculous exudates in pneumothorax, thoracoplasty and phrenic paralysis cases demonstrates the active part played by the normal elastic lungs and normally functioning bronchial tree in accomplishing adequate drainage. Upon this depends, not only the prevention of the development and spread of tuberculosis, but the clearing of an already existing lesion.

## THE EFFECTS OF PATHOLOGICAL CHANGES WITHIN THE PARENCHYMA OR WITHIN THE INTERSTITIAL PORTION OF THE LUNG ON THE DRAINAGE MECHANISM

The effects of exudative changes within the parenchyma of the lung on drainage of exudate from the bronchial tree have already been discussed. In general, it is believed that any pathological process—exudative, infiltrative or fibrotic—within the lung, which has a tendency to limit its elasticity, would favor the retention of tuberculous exudates and, therefore, would favor a chronic and progressive disease. In like manner, pathological changes within the interstitial portion of the lungs along the walls of the bronchi, such as are seen in silicosis and perhaps also in old age, would provide a fertile field for the implantation of tubercle bacilli and a retention of them and their exudates within the bronchial tree. This would, therefore, favor the development of tuberculosis in such individuals. Some of the manifestations of silicosis are loss of elasticity of the lungs due to peribronchial beading and thickening with fibrosis. This may be slight at first, but may become marked later as the

disease advances. Pleural adhesions may also alter the normal "peristaltoid" movements of the bronchial tree. The diaphragm in late stages of silicosis may become tented and there may be a marked limitation of motion of the diaphragm. All of this would favor a stagnation of exudate within the bronchial tree.

It has long been known that secondary bacterial invasion of the silicotic lung is not uncommon and according to Gardner,[9] seventy-five per cent of those in whom silicosis develops die of tuberculosis. Chronic bronchitis, bronchiectasis, abscess and pneumonia are not uncommon complications. This susceptibility to bacterial infection has not been adequately explained. Irritation of the respiratory tissues by the silica particles rendering the mucous membranes more susceptible has been advanced as one explanation. The toxic effect of certain organic dusts has been suggested as an influencing factor. It is believed, however, that the chief factor influencing the development of tuberculosis in individuals suffering with silicosis is a mechanical one. The ciliary movements, the coughing reflex, and the lengthening and shortening movements of the bronchial tree are able, under normal conditions, to cleanse the lungs and bronchial tree of accumulated infectious exudates. When the normal elasticity of the lungs is impaired, as in silicosis, exudates within the bronchial tree cannot be evacuated effectively and disease will result. Ordinarily, small numbers of tubercle bacilli inhaled into the lungs of individuals already primarily infected produce a relatively small amount of exudate and may, therefore, be easily evacuated through this mechanism described above. In a hardened silicotic lung, however, small numbers of bacilli inhaled into the lungs may be retained and set up a reinfection tuberculosis which is usually fatal. For this reason, it is my belief that exogenous reinfection plays a leading role in the development of progressive tuberculosis in individuals with silicosis. Cummings[10] has "demonstrated for example that it is perfectly possible to have an excessive silicosis hazard without an associated increase in the frequency or severity of tuberculosis, and, conversely, an excessive rate and degree of tuberculosis with only a mild silicosis hazard. In one mine of 1200 where silicosis was a serious problem not one case of tuberculosis was found." This is explained on the absence of infection in the community, and emphasizes the point that while silicosis predisposes to tuberculosis, it is necessary to have sources of infection if the disease becomes prevalent. He also showed that fresh tuberculosis occurs in all ages in silicosis patients and that new infections were not uncommon after 40.

It is interesting to note that anatomically the distribution of the lesion in silicosis corresponds to a certain extent, in the early stages at least, to that of primary tuberculosis. The silicotic lesions may

extend into the apices but in a large number of early cases the apices are not involved. As is well known, the primary tubercle is found more commonly in the lower part of the upper lobe or upper part of the lower lobe. It is believed that this is not a mere coincidence, but is due to the fact that tubercle bacilli and dust particles are carried more directly by the air current to the areas mentioned. Tubercle formation in primary infection develops in that portion of the lung in which the bacilli first lodge. They are phagocytized and carried, like the dust particles, to the nearest lymphoid tissue within the interstices of the lung. The tissues are not allergic to them and they do not remain within the bronchial tree as do bacilli of reinfection. The evacuation mechanism of the bronchial tree does not play an important part, therefore, in the prevention of primary tuberculosis for reasons stated above.

This brings us to a discussion of tuberculosis in the aged and why it becomes prevalent in this age group.

There are no obvious reasons, according to Cummings[10] and others, for expecting a different physiological response to the tubercle bacillus before the age of 40 than after this age, and there are no very good reasons to believe that there is an alteration of the immune mechanisms in the aged. X-ray films of old individuals show rather characteristically definite thickening along the bronchial tree. Frequently, too, thickening of the pleura may be seen. Calcification of the cartilages is the rule in the aged and this factor, together with a loss of resilience of the lungs, favors the implantation and retention of bacteria in the bronchial tree. Again, as in silicosis, the bronchial tree finds it more difficult to cleanse itself of exudates with the result that pulmonary disease is a frequent complication. The high mortality rate from tuberculosis in old men may be explained by the fact that they are exposed to a greater extent than women in the same age group. H. L. Sampson[11] interpreted a series of 774 films taken on Kentucky coal miners, and the results of this study seem to show that reinfection may develop in any age period, more particularly late in life. It was found that there was an increase in frequency of tuberculous lesions in each successive five-year age period.

Sampson[11] points to a very significant fact that in following 8000 miners for an eight-year period none of them showed at any time during this period new tuberculosis developing in the immediate vicinity of a calcified primary focus. This would favor the view that exogenous reinfection occurs often. It also favors the view that tubercle bacilli and their exudates which would ordinarily be evacuated from the normally functioning bronchial tree would be retained in the aged due to a loss of elasticity of the lungs. A lack of immunity does not explain the increased morbidity and mortality

in the aged. Let us always keep in mind stenotic endobronchial tuberculosis and its sequelae and we can never minimize the importance of drainage in every tuberculous individual, no matter what may be his race or age.

## SUMMARY

1) The normal bronchial evacuation mechanism is briefly reviewed.

2) This normal bronchial mechanism may be truly as effective in protecting the individual from tuberculous reinfection of exogenous origin as the lymphatic system is effective in preventing reinfection from primary foci situated within the lung.

3) The mechanical failure of the bronchial tree to cleanse itself of infectious exudates plays a leading role in the pathogenesis of pulmonary tuberculosis.

4) Certain factors which might have a definite influence on the retention of infectious exudates within the bronchial tree are discussed as follows:

(a) Physical obstruction and developmental abnormalities.

(b) Degree of allergic response to reinfection.

(c) A difference in functional ability of the bronchial tree in apex and base of the lung in determining localization of pulmonary tuberculosis.

(d) Alteration of the normal functions of the bronchial tree following collapse therapy and its effects on the drainage mechanism.

(e) The effects of pathological changes within the parenchyma or within the interstitial portion of the lung on the evacuation mechanism.

5) Exogenous reinfection tuberculosis is no doubt much more common in all age groups than generally believed.

## RESUMEN

1) Se repasa suscintamente el mecanismo normal de la evacuación bronquial.

2) Este mecanismo bronquial normal puede ser verdaderamente tan eficaz en proteger al individuo contra la reinfección tuberculosa de origen exógeno como el sistema linfático es eficaz en prevenir la reinfección que origina en focos primarios situados dentro del pulmón.

3) El fracaso mecánico del árbol bronquial en su función de depurarse de los exudados infecciosos desempeña un papel prominente en la patogenia de la tuberculosis pulmonar.

4) Se discuten ciertos factores que pueden ejercer una influencia bien definida en la retención de exudados infecciosos en el árbol bronquial. Estos son:

(a) Obstrucción física y anomalías en el desarrollo.

(b) El grado de la reacción alérgica a la reinfección.

(c) La diferencia en la capacidad funcional del árbol bronquial en el vértice y en la base del pulmón y su efecto sobre la localización de la tuberculosis pulmonar.

(d) Alteración en la función normal del árbol bronquial consecutiva a la colapsoterapia y sus efectos sobre el mecanismo de drenaje.

(e) Los efectos de las alteraciones patológicas en el parénquima o en la parte intersticial del pulmón sobre el mecanismo de evacuación.

5) La tuberculosis de reinfección exógena es indudablemente mucho más común en todos los períodos etarios de lo que generalmente se supone.

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# The Rationale of the Therapeutic Administration of Massive Doses of Vitamin A in Tuberculous Diabetics*

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The incidence of tuberculosis in diabetics is higher than in the general population. We[1] have presented previously a composite tabulation of diabetics from the reports of seven clinicians representing 8,520 patients. It was pointed out that tuberculosis was found in 2.6 per cent in this group; this figure was three times the corresponding percentage in the general population. Since then Lyon[2] reported that tuberculosis was the cause of death in 7.3 per cent of 150 diabetic deaths; while the roentgenologic studies of Kramer and Lawson[3] revealed an incidence of 1.2 per cent in 408 diabetics. Ralli and Steinberg[4] observed 748 diabetic patients during a seven-year period. Of these 33 had active pulmonary tuberculosis, an incidence of 4.3 per cent. Root[5] investigated this problem in a large group of diabetic patients. He found an incidence of 2.8 per cent in adults; furthermore, he pointed out that reinfection type of pulmonary tuberculosis was more than 13 times as frequent in children who developed diabetes before the age of 15 years as among Massachusetts school children, and 16 times as frequent in adolescent diabetics who developed the disease between the ages of 15.1 and 19.9 years as in a corresponding group of high school students. As to the higher age groups Root quotes the figures of Bolduan[6] who found that the incidence of tuberculosis in persons who died at the age of 45 years and over was 4.4 per thousand males and 1.3 per thousand females. The expected calculated incidence of pulmonary tuberculosis was 2.5 males and 1.5 females per thousand; however, the average recorded incidence of pulmonary tuberculosis among the diabetes deaths for the same two-year period of 1930 and 1931 was among males 18.5 and among females 23 per thousand. Wiener and Kavee[7] reported an incidence of 23.9 per cent in 909 diabetics admitted to Montefiore Hospital in New York City; this high figure was explained on the basis that a large proportion of the hospital census was made up of tuberculous patients. A report by Escudero[8] from Argentina showed the presence of active pulmonary tuberculosis in 9.1 per cent in a group of 735 diabetic patients. Of the

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European observers Blanco Soler[9] recorded a frequency of only 1 per cent; while Pilgerstorfer[10] saw 71 cases of pulmonary tuberculosis, or 5.9 per cent, in 1208 diabetics. A study of 1441 diabetics by Boller[11] revealed that the frequency of pulmonary tuberculosis was 10.17 per cent in males and 6.16 per cent in females. Valdes Ruiz[12] found tuberculosis in 2.8 per cent of his diabetic patients. In a group of 143 hospital and insurance cases of Lorenzen[13] pulmonary tuberculosis complicated diabetes in 5.5 per cent.

Two series of experiments of Steinbach and his associates[14] brought objective confirmation of the opinion that there is an increased susceptibility to tuberculosis in diabetics. They inoculated dogs with a pathogenic bovine strain of tubercle bacilli and found that, while the controls developed a limited tuberculosis in the kidney or in the spleen, the tuberculous process in the pancreatectomized diabetic animals was more invasive, involving more organs. In another pertinent experiment albino rats were rendered hyperglycemic by pancreatectomy and by subsequent repeated intraperitoneal injections of extract of the anterior pituitary gland. From their observations they were able to ascertain that the natural resistance of the rat could be lowered and that tuberculosis developed more extensively in the hyperglycemic animal. Also, they noted that the tubercles were more conglomerate and widespread, and contained many more tubercle bacilli than those of the controls.

The cause of predisposition of diabetics to tuberculosis is a much debated and as yet apparently an unsolved problem. The oldest theory is that hyperglycemia favors the growth of tubercle bacilli in the body and therefore it is primarily responsible for the increased susceptibility of diabetics. However, the experimental studies of Richardson,[15] Pillsbury and Kulchar,[16] Marble, White and Fernald[17] revealed that the increased sugar content of the blood and tissues is not the causative factors in lowering the resistance of the body or aiding the propagation of infectious microorganisms in the tissues. The recently formulated authoritative opinion of Mosenthal and Mark[18] relegates this traditional concept to oblivion. They state emphatically that the elevation of blood sugar in the absence of glycosuria is not only harmless but even beneficial, because hyperglycemia promotes the utilization of sugar, does not cause degenerative changes in the tissues, and does not predispose to infections. This opinion is in harmony with that expressed by Keeton[19] who pointed out that wound healing advances rapidly in the presence of hyperglycemia when insulin is given in quantities inadequate to control it. According to Keeton, a much more plausible explanation is a local acidosis with its disturbed electrolyte balance which interferes with the water transport. He also calls attention to the fact that even in controlled cases there is an

absence of defensive tissue reaction to staphylococcus albus, a normal inhabitant of the skin. The latter statement is supported by the experimental investigations of Horster.[20] He found that in dogs rendered diabetic by pancreatectomy smaller numbers of staphylococci are sufficient to produce infection than in the controls and that the abscess formation at the site of staphylococcus infection was more intense in the diabetic dogs than in the control animals. Furthermore, he ascertained that the complement and hemolysin titers and the formation of agglutinin do not differ from that in the controls. Grafe[21] quotes daCosta and Beardley who observed that the opsonic index in severe diabetics is lower than normal. Also, the studies of Moen and Reimann[22] indicate a lowering of the opsonic index, the complement, antibody production and of the bacteriostatic capacity of the blood in diabetic patients. Horster[20] found that the leukocytes of the diabetic organism showed a loss in phagocytic power as compared with normal white blood cells. Long[23] expressed the opinion that the cause of predisposition may lie in the diabetic patient's disordered fat metabolism through an increased availability of glycerol which is one of the best nutrients for acid fast microorganisms. Root[5] pointed out that the growth of tubercle bacilli in the human body depends upon chemical factors, such as accessibility of substances for nutrition, the reaction and oxygen tension of the tissues, water relations, and other so far unexplored factors. The fact that diabetic coma and uncontrolled diabetes were followed frequently by a flare-up was thought to be partly attributable to an oversupply of nitrogenous compounds that help to support the growth of tubercle bacilli. Furthermore, Root[5] emphasized the possible importance of changes in the reticulo-endothelial system. It is known that interference with the normal function of this system causes a decrease in the defense and resistance of the body against infection. In diabetes the reticulo-endothelial cells are sometimes found to be distended with fat, especially when diabetes is relatively uncontrolled; consequently their normal defensive function is lowered. Keeton's[19] conception that the appearance of acidosis destroys the resistance of the tissues and favors the spread of the tuberculous process is in line with the experience of Smithburn[24] who demonstrated in his experimental studies that one can increase the virulence of the tubercle bacilli by increasing the acidity of the culture medium. The impaired resistance of diabetics to infection is considered by Moolten[25] a part of the general vulnerability of the diabetic tissues to injury of any type and is attributed to deficient cellular oxidation.

From the study of the available clinical and experimental investigations one gains the impression that the role of vitamin A may explain in a large measure the increased susceptibility of the dia-

betic to tuberculosis. Vitamin A deficiency causes specific pathologic changes in the respiratory mucosa. These consist of atrophy of the epithelium with associated disappearance of the ciliary function, proliferation of the basal cells, and replacement of the original epithelium by a stratified keratinizing epithelium. Bessey and Wolbach[26] state that in the lungs of human infants as well as of experimental animals these changes lead to the occlusion of bronchi, formation and filling of bronchiectatic cavities with keratinized cells, and atelectasis; the plugs of desquamated epithelial cells act as a culture medium for pathogenic microorganisms. They assume that the frequency of pneumonia in infants may be explained on the basis of vitamin A deficiency as one of the underlying causes. The pneumonia is invited by the atrophy of the mucosa that precedes the appearance of keratinizing epithelium; the pneumonia is usually of the interstitial and peribronchial type. Wilson and DuBois[27] made pathologic studies of a fatal case of nutritional keratomalacia in an infant, and noted an extensive keratinization of the bronchial epithelium, bronchiectatic cavities and abscesses. Pneumonia was the immediate cause of death in two of four cases of Ross[28] in whom keratomalacia was diagnosed during life. Of eighty-six children with xerophthalmia observed by Bloch[29] pneumonia was detected in fifteen and bronchitis in twelve.

While it is generally accepted that resistance to infection in man may be decreased in cases with marked deficiency of vitamin A, numerous contradictory communications have been published concerning the antiinfectious property of this vitamin since 1922 when vitamins A and D were identified and separated by McCollum and his associates.[30] Concerning this problem the Council on Pharmacy and Chemistry of the American Medical Association[31] arrived at the conclusion in 1939 that vitamin A is not a specific in the prevention of colds, influenza and such infections and that it has not been demonstrated that the ingestion of vitamin A far in excess of that necessary for normal body function and readily obtained from a properly selected diet is an aid in preventing various types of infection. Furthermore, it was stated that "the present indications are that vitamin A is an aid toward the establishing resistance of the body to infections in general only when there has been a decrease of body reserves and the ingestion of vitamin A is inadequate."

The occurrence of vitamin A deficiency in diabetes was reported in the recent medical literature. Brazer and Curtis[32] observed twenty patients with juvenile diabetes mellitus and found evidence of hypovitaminosis A in all of them by the Frober-Faybor biophotometric method. They were able to correct this condition by the daily administration of 60,000 U.S.P. units of vitamin A. Carotene that is

present in the customary diet is the source of vitamin A and it is converted to vitamin A by the liver. Brazer and Curtis maintain that in diabetics the apparent cause of the hemeralopia is the inability of the liver to convert carotene to vitamin A. This opinion coincides with that expressed earlier by Ralli, Brandaleone and Mandelbaum.[33] Monceaux[34] recommended the administration of vitamin A in preference to carotene to patients with liver disease, in general, for the reason that there is deficient transformation of carotene to vitamin A in hepatic diseases. Freston and Loughlin[35] studied a group of juvenile diabetics, from 7 to 19 years of age. The duration of diabetes in the majority of the children was one to five years. In none of the 33 chemical determinations on 24 of the children was the carotene low. Of seven patients rated as poorly controlled, 71 per cent were low in vitamin A. Of seventeen patients excellently or well controlled, 48 per cent were low in vitamin A. White and Gordon[36] reported that in diabetics the blood carotene values varied between 5 and 25 dichromate units as compared with 2 and 8 in the normal. Schroeder[37] pointed out that carotene as well as vitamin A disappeared from the blood during diabetic coma. It is known that normally about 95 per cent of the body's vitamin A content is stored in the liver. In diabetes this is reduced. According to the report of Popper and Greenberg[38] who analyzed the vitamin A content of the lung with the aid of fluorescence microscopy, the normal lung contained small quantities of vitamin A in the alveolar septa, apparently in the capillary epithelium and the interstitial cells; also accumulations were seen in the perivascular tissue and in the subserosa of the pleura; but in hypovitaminosis and avitaminosis the lung was free of vitamin A. If hypovitaminosis A in diabetics entails the total disappearance of vitamin A from the lung, it seems possible that such condition may lower the resistance of this organ and consequently may facilitate the settling and propagation of tubercle bacilli, particularly because of the lack of replacement possibilities due to the depletion of the vitamin A depots in the liver and to the low level of vitamin A in the blood.

Commenting on the importance of the glycogen of the liver, Roger[39] pointed out that almost all chemical activities of the liver depend on the integrity of its glycogen function. Schneider and Widman[40] called attention to the fact that the vitamin A metabolism is closely coupled with the glycogen metabolism of the liver. It is known that in uncontrolled diabetes the glycogen disappears from its normal depots in this organ. This circumstance, then, explains the low vitamin A values found in the liver of diabetics. If the capacity of the liver to convert carotene (provitamin A) into vitamin A is diminished in diabetes, as has been pointed out by several investigators, a diet containing apparently adequate amounts

of carotene must be ineffective for restoring the normal vitamin A content of the liver and of the blood. Brazer and Curtis[32] found that the daily administration of crystalline carotene, dissolved in vegetable oil, corresponding to 60,000 U.S.P. units of vitamin A, for as long as fourteen days, did not affect the light adaptation of their diabetic patients. Confirmatory evidence of the inability of the diabetic organism to convert carotene to vitamin A was demonstrated by Ralli, Pariente, Brandaleone and Davidson[41] and by Brandaleone and Ralli.[42] They found that the average fasting blood carotene in a group of diabetic patients was 0.262 mg. per hundred cubic centimeters and in a group of normal subjects was 0.109 mg. Also they studied the effects of the daily oral administration of 0.3 per cent carotene in oil on the blood carotene of normal and diabetic individuals for periods varying from one to four months. Following the administration of 1 cc. of carotene solution there was a greater increase in the blood carotene, and a more gradual return to the fasting level in diabetic patients than in the controls. Following the administration of 5 cc. of carotene solution to one diabetic patient and to a normal subject whose blood carotene level had been elevated as the result of the previous administration of a large dose of carotene, there was a still greater increase in the blood carotene in the diabetic patient above that of the normal subject and clinical evidence of carotenemia appeared.

If, from the analysis of these data, it is correct to formulate the premise that in diabetes hypovitaminosis A occurs frequently, and—because of the diminished competency of the liver to convert carotene (provitamin A) to vitamin A—the ingested carotene (provitamin A), taken in sufficient amounts in the daily diet, is not adequately utilized, it is logical to conclude that in this hepatopancreatic dysfunction lies the main conditioning factor for the increased susceptibility of diabetics to tuberculosis.

The circumstance that the diabetic is often in a state of subclinical vitamin deficiency signifies a twofold therapeutic implication: (1) the correction of the carbohydrate metabolism by insulin (the absence of insulin prevents the storage of glycogen in the liver); and (2) replacement therapy with massive doses of vitamin A.

The rationale of the administration of massive doses of vitamin A to tuberculous diabetic patients is well supported by a number of competent observations. Keeping in mind the parallelism between the glycogen metabolism and vitamin A metabolism in the liver, the pertinent research work of Rabuchin[43] is of interest. Groups of guinea pigs and rabbits were rendered tuberculous by inoculation. The postmortem examination of these animals dead of tuberculosis revealed low glycogen content in the liver. The latter may have

been due to increased metabolism caused by the absorption of toxins or to a shortage of insulin. Studies of the adrenalin-glucose curve, after the intravenous injection of 0.02 mgm. or the subcutaneous administration of 0.7 mgm. of adrenalin was marked by a lesser increase in the blood surgar in patients with extensive, progressive tuberculosis than in healthy individuals. These findings point to a possible anatomical damage of the liver as emphasized by Kugelman[44] who found a low adrenalin-glucose index in patients with severe parenchymal lesions of the liver. Ritzman's[45] opinion that the adrenalin-glucose curve is proportionate with the glycogen content of the liver is generally accepted, therefore a flat curve in advanced tuberculosis may be taken as suggestive of low liver glycogen. Furthermore, Rabuchin[43] noted that amylase, which splits glycogen into maltose that, in turn, is transformed into two molecules of dextrose by maltase, is increased in the blood of tuberculous patients, particularly in far-advanced decompensated cases.

It can be readily seen that when diabetes is complicated by a severe, far-advanced pulmonary tuberculosis one is bound to find a significant degree of hepatic dysfunction. Crimm and Short[46] examined the livers of fifty patients who died of pulmonary tuberculosis. They found that the vitamin A content of the liver approached depletion in 14 per cent of the patients. Tuberculous patients with temperatures ranging over 100 degrees F. had reductions of approximately 50 per cent in the vitamin A content of the liver compared to tuberculous patients with a temperature course below 100 degrees F. who showed nearly normal levels of hepatic vitamin A. The clinical study of Raimondi and d'Amato[47] indicates that hepatic insufficiency is commonly observed in patients having pulmonary tuberculosis. They state that there is a parallelism between the activity of the tuberculous lesion and the competency of the liver function. Also, the occurrence of hypoprothrombinemia in pulmonary tuberculosis suggests liver damage. It has been demonstrated by Andrus and Lord[48] that a normally functioning liver is necessary for the maintenance of a normal level of prothrombin in the blood. Keeping in mind the possible influence of low vitamin K intake and diminished vitamin K absorption from the intestines in this disease, the observations made by Sheely[49] are of importance. Among his 106 cases of active and chronic pulmonary tuberculosis studied in regard to concentration of prothrombin in the blood, a significant deficiency of prothrombin was found in 51. Seventy-three per cent with minimal, 60 per cent with moderately advanced and only 9.6 per cent of the far-advanced tuberculosis had a prothrombin concentration within normal range. He concluded that the prothrombin concentration in the blood of tuberculous patients seemed to be related to the toxemia due to the tuberculous disease in the lungs.

Levy[50] found that of his patients with pulmonary tuberculosis 32 per cent had more or less marked degree of hypoprothrombinemia, while there was no evidence of this condition in the control group. Also, he observed parenchymal liver damage in 87 per cent of patients with pulmonary tuberculosis, while 33 per cent of the control subjects showed a slight degree of liver dysfunction.

Wolf[51] carried out studies on the vitamin A content of the blood in tuberculosis and found that in the majority of patients with open pulmonary tuberculosis the vitamin A values of the blood were noticeably reduced. Vitamin A deficiency in tuberculosis was investigated by Getz and his associates[52] by the biophotometric method. Their findings revealed that pathologic deficiency was present in 37.5 per cent of the primary, 57.6 per cent of the minimal, 47 per cent of the moderately advanced and 60.7 per cent of the far-advanced cases of pulmonary tuberculosis. Subsequently Getz and Koerner[53] made determinations of the vitamin A content in the blood plasma by photoelectric colorimeter. For comparison, the values found by Kimble[54] and by Scalongne[55] were used, namely, normal: 110 I. U. and above for male and 95 I. U. and above for female; borderline: 85-110 for male and 75-95 for female; the corresponding figure indicative of pathologic deficiency are below 85 and below 75, respectively. They stated that the level of vitamin A in the plasma of recently diagnosed tuberculous patients was lowered in proportion to the extent of the tuberculous involvement.

Another possible source of the impaired vitamin A metabolism was reported by Breese, Watkins and McCoord.[56] They studied the utilization of vitamin A in patients with active pulmonary tuberculosis and intestinal symptoms. They noted that following the administration of 7000 U.S.P. units of vitamin A per kilogram of body weight the mean maximum rise in vitamin A in the blood of normal persons was approximately twice that of all patients with tuberculosis. Furthermore, the blood of patients with mild intestinal symptoms reached, on the average, twice the level of those with moderate and severe symptoms. They offer the opinion for the explanation of these findings that there is a functional derangement of the absorption of vitamin A in tuberculous patients. They thought that actual tuberculous disease of the intestine, liver or pancreas probably was not a factor, for in the majority of patients who came to postmortem examination no gross or microscopic lesions were found in these organs.

It is reasonable to conclude from these data and from the findings that indicate the occurrence of hepatic dysfunction in severe forms of pulmonary tuberculosis that the administration of massive doses of vitamin A, from 150,000 to 200,000 units daily, may be a valuable supplementary therapeutic measure in the management of certain types of pulmonary tuberculosis.

## CONCLUSIONS

1) It is the general consensus that diabetes mellitus predisposes to tuberculosis.

2) So far, no uniform understanding has been reached as to the specific cause or causes of this predisposition.

3) It is known that there is an increased susceptibility to infections in individuals in whom there is a considerable degree of vitamin A deficiency and whose food intake is inadequate.

4) Experimental and clinical investigations revealed that in diabetes mellitus there is frequently a state of subclinical vitamin A deficiency. This is brought about by the diminished competency of the liver to convert carotene (provitamin A)—as supplied in the daily diet—to vitamin A.

5) There is experimental and clinical evidence that tuberculosis has an untoward effect on the glycogen metabolism of the liver and consequently on the vitamin A storage and exchange that are closely associated with the glycogen balance of this organ.

6) Dietary increase in the carotene (provitamin A) intake is not likely to be of value for correcting hypovitaminosis A because of the hepatopancreatic dysfunction in diabetes mellitus.

7) Functional derangement of the intestinal absorption of vitamin A in tuberculosis reduces considerably the effectiveness of the generally prescribed doses of vitamin A.

8) Although we are aware of the possible influence of other potential predisposing factors, it is our opinion that vitamin A deficiency is one of the main causes of the increased susceptibility of the diabetic to tuberculosis.

9) In view of the fact that diabetes mellitus as well as a high percentage of the moderately and far-advanced stages of pulmonary tuberculosis are conducive to vitamin A deficiency, partly because of insufficient intestinal absorption of this vitamin and partly because of the diminished competency of the liver to convert carotene (provitamin A) to vitamin A, it is our opinion that the administration of massive doses of vitamin A, from 150,000 to 200,000 units daily, may serve as a useful adjunct in the treatment of pulmonary tuberculosis and particularly in the management of diabetes mellitus complicated by pulmonary tuberculosis.

## CONCLUSIONES

1) El consenso general de opiniones es que la diabetes mellitus predispone a la tuberculosis.

2) Hasta ahora no se ha llegado a un acuerdo uniforme acerca de la causa o causas específicas de esta predisposición.

3) Se sabe que son más susceptibles a las infecciones los sujetos en quienes existe un grado considerable de deficiencia de vitamina A y cuyo régimen alimenticio es inadecuado.

4) Investigaciones experimentales y clínicas revelan que en la diabetes mellitus existe con frecuencia un estado de deficiencia subclínica de vitamina A. Esto se debe a disminución de la capacidad del hígado para convertir *carotene* (provitamina A)—provista en los alimentos diarios—en vitamina A.

5) Existen pruebas experimentales y clínicas de que la tuberculosis tiene un efecto desfavorable sobre el metabolismo del glucógeno en el hígado y, por consiguiente, sobre la fijación y el intercambio de vitamina A que están íntimamente relacionados con el equilibrio del gucógeno en este órgano.

6) No es probable que el aumento de *carotene* (provitamina A) en la dieta ayude a corregir la hipovitaminosis A, debido a la disfunción hépato-pancreática presente en la diabetes mellitus.

7) El desorden funcional en la absorción intestinal de vitamina A en la tuberculosis disminuye considerablemente la eficacia de las dosis de vitamina A que generalmente se recetan.

8) Aunque estamos enterados del posible efecto de otras causas potenciales de esta predisposición, opinamos que deficiencia en vitamina A es una de las causas principales de la mayor susceptibilidad del diabético a la tuberculosis.

9) En vista del hecho de que tanto la diabetes mellitus como un alto porcentaje de los períodos moderadamente avanzados y muy avanzados de tuberculosis pulmonar tienden a causar deficiencia en vitamina A, debida en parte a la absorción insuficiente de esta vitamina en los intestinos y en parte a la disminución de la capacidad del hígado para convertir *carotene* (provitamina A) en vitamina A, opinamos que la administración de dosis masivas de vitamina A, de 150,000 a 200,000 unidades diarias, puede ser un coadyuvante provechoso en el tratamiento de la tuberculosis pulmonar y, particularmente, en el tratamiento de la diabetes mellitus complicada por tuberculosis pulmonar.

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# Tuberculous Meningitis Complicating Thoracic Surgery*

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Tuberculous meningitis is not rare, but it is one of the more unusual complications of the reinfection type of pulmonary tuberculosis. Consequently the relatively high incidence of this condition among the deaths of our surgical patients has attracted our attention. Although meningitis is mentioned as a rather frequent seqence of genito-urinary surgery there are no reports in the literature describing its relationship to thoracic operations other than a previous communication by the author.[1] In this earlier article cases are reported in which the disease developed following an intrapleural pneumonolysis and also after a second-stage thoracoplasty.

In the past year and a half 250 patients have had chest surgery in this institution. Six of these individuals eventually died within this period, three of them from tuberculous meningitis.

The first case (5725) was a man of thirty-eight who had a three-stage, eight-rib thoracoplasty after eight months of other treatment. He had a fibro-ulcerative lesion involving the left lung, and stabile scattered deposits in the right. The man seemed to be a good surgical risk except for two conditions: He worried about himself to the point where he developed a definite anxiety neurosis; and his teeth were carious and had to be removed. However, his temperature was normal and he had no neurologic signs or symptoms.

Convalescence following the first two stages was entirely uneventful but the febrile reaction failed to subside in the usual manner after the last operation. On the fourth postoperative day the patient began to complain of severe, persistent headache and vomiting. A large carbuncle developed on the back some distance from the operative region. Examination of the pus from the carbuncle revealed staphylococci and sulfathiazole was given for one week. The situation went from bad to worse and the patient became irrational on the thirteenth day, finally stuporous, and died seventeen days postoperatively. A postmortem spinal tap yielded fluid which contained, after standing, a fibrin web and numerous tubercle bacilli.

The second patient (5730) had a four-stage, eight-rib thoracoplasty for a fibro-ulcerative lesion in the upper half of the left lung after seven months previous treatment proved ineffective. There were scattered stabile deposits throughout the remainder of the lung field and a tuberculous bronchitis of the granular type. However, the general condition was quite good and there was no evidence of extra-pulmonary disease.

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The postoperative course following the first three operations was satisfactory. The temperature returned to normal three days after the last, an anterior stage, only to rise again ten days subsequently. Coincident with the development of fever, the man began to complain of headaches and vomiting. Two weeks later he became mentally confused, his reflexes were increased, Kernig's sign was positive and his neck became rigid. He died one month after the last operation. The spinal fluid was examined twice before death. The cell count rose from 55 at the first examination to 130 the day he died. Globulin was 0, albumin three plus, and no tubercle bacilli were found.

The third case (5686) was a male negro, age 36, who had a left phrenemphraxis as a substitute for pneumothorax which was lost prematurely due to obliterative pleuritis. At the time of the operation he had a bilateral lesion of the hematogenous type with small cavities in the left apex. He had been under treatment for six months, which resulted in general and local improvement. There were no symptoms referable to extra-pulmonary disease. A low-grade fever developed about three weeks after the operation, followed by persistent headaches one month later. Shortly after the appearance of headaches, the patient became mentally confused and the neurologic signs were positive. A culture of the spinal fluid collected shortly before death revealed tubercle bacilli. The man died eleven weeks postoperatively, two months after the onset of symptoms.

## CONCLUSIONS

Specific meningeal involvement was proved bacteriologically in two of these three cases, and evidence was sufficiently clear in the other patient to justify a reasonably certain clinical diagnosis. The relationship of the complication to the operations is of course difficult to prove. However, there was no indication of the presence of a complication in any of these cases before operation. In fact, all of the patients seemed to be fairly good surgical risks. There is only one characteristic common to all three cases: Each had scattered deposits in the contra-lateral lung of the hematogenous type which were judged to be stabile after observation for several months.

The incubation period of tuberculous meningitis is somewhat indefinite, but the onset of symptoms in each case was within the arbitrary time limits usually set for the development of complications from thoracic surgery.[2]

The pathogenesis of the meningeal condition must have occurred in one of the following ways:

1) A focal reaction of a latent, previously unrecognized cerebral-spinal focus may have occurred, but there is no evidence in any of our cases to support this theory of infection of these areas prior to surgery.

2) Autotuberculinization of the pulmonary lesion may have taken place with subsequent spread into a pulmonary vein. X-ray films taken two to four weeks after operation did not show the uniform haze usually associated with focal reactions of pulmonary lesions.

3) Emboli composed of tubercle bacilli may have been liberated into the circulation during the operation. The chest wall was sound in each case, so no infected tissues were entered during surgery. It seems most likely that collapse of the diseased area of the lung disturbed the protective organization of the tissues which resulted in the discharge of organisms into the circulation where they were carried to the small vessels of the meninges.

Emphasis is usually placed on bronchial spread as a complication of thoracic surgery.[2] However, our experience leads us to believe that hematogenous dissemination happens almost as often. Tuberculous meningitis has accounted for half of the deaths of our surgical patients.

## CONCLUSIONES

La invasión meníngea específica fue comprobada por medio de exámenes bacteriológicos en dos de los tres casos, y en el otro paciente los signos fueron lo suficientemente claros para justificar el diagnóstico clínico razonablemente acertado. La conexión entre la complicación y las operaciones es, por supuesto, difícil de comprobar. Sin embargo, no hubo indicación alguna de que la complicación existiera en ninguno de estos casos antes de la operación. En efecto, todos los pacientes parecían ser riesgos quirúrgicos razonablemente buenos. Hubo sólo una característica común a los tres casos: Cada uno tenía en el pulmón contralateral depósitos esparcidos de tipo hematógeno que se juzgaron ser estables después de varios meses de observación.

El período de incubación de la meningitis tuberculosa es un tanto indefinido, pero la iniciación de los síntomas en cada caso tuvo lugar dentro del término arbitrario que se suele fijar para la producción de las complicaciones de la cirugía torácica.[2]

La patogenia del estado meníngeo debe de haber ocurrido en una de las maneras siguientes:

1) Puede haber sobrevenido una reacción focal en un foco cerebroespinal latente, no reconocido previamente, pero no se observaron signos en ninguno de nuestros casos para sostener esta teoría de infección de estas zonas antes de la operación.

2) Puede haber tenido lugar autotuberculinización de la lesión pulmonar con propagación subsecuente a una vena pulmonar. Las películas radiográficas tomadas de dos a cuatro semanas después de la operación no manifestaron la opacidad uniforme que se suele observar en reacciones focales de lesiones pulmonares.

3) Es posible que embolias compuestas de bacilos tuberculosos hayan entrado a la circulación durante la operación. La pared torácica estaba sana en cada caso, así es que no se invadió ningún tejido infectado durante la operación. Parece ser más probable que

el colapso de la zona afectada del pulmón alteró la organización protectiva de los tejidos, lo cual resultó en la entrada de gérmenes a la circulación la que los condujo a los pequeños vasos meníngeos.

Se suele hacer hincapié sobre la propagación broncógena como complicación de la cirugía torácica.[2] Sin embargo, nuestra experiencia nos hace creer que la diseminación hematógena sucede con casi tanta frecuencia. La meningitis tuberculosa ha causado la mitad de las muertes en nuestros pacientes quirúrgicos.

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# Tuberculosis in Canada

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At war since September, 1939, Canada, now in the fifth year, is closely approximating total national effort. How does this affect the tuberculosis situation of today? Certainly less adversely, we believe, than was the case a quarter of a century ago. In the first World War Canada accepted for military service 619,636 men without the benefit of a single x-ray. It is not known how many of this number had tuberculosis on enlistment or on discharge from service. We do know, however, that, in the eighteen-year period following the Armistice, of all deaths (15,576) occurring in Canadian soldiers *pensioned* for any disability, 23 per cent were due to tuberculosis. In the 7,530 pensioners in whom the disease resulting in death was considered a direct service disability, 39 per cent died from tuberculosis. The percentage rose to 51 in those of this group whose service was in Canada only. F. S. Burke points out the very significant fact that the average age of tuberculosis pensioners was two years lower than that of the whole group of pensioners. In his publication, "Deaths Among War Pensioners," Burke's calculation of the mortality rates for Class One of the International List (88% tuberculosis) is enlightening and startling. At 20 years of age the rate per 100,000 for Canadian pensioners at the end of the last war was at the extremely high figure of 880 as compared to 460 at 30 years and 276 at 40 years. The comparable figure for the 20-year-old men in the Canadian population of 1930 and 1932 was 120. Thus is evident the disastrous effect of the combination of youth, tuberculosis, and military service.

The gravity of the problem was not fully appreciated before 1917, which year saw the first large groups of tuberculous soldiers invalided from overseas. These returned to a Canada not then too well equipped to receive and treat them adequately. Almost overnight, however, sanatoria, aided by Federal grants, undertook an extensive building program and the development of an enlarged, trained staff. Thus, before World War I was concluded Canada had become tuberculosis conscious to a degree that a decade or two of educational effort previously had failed to achieve. The momentum then developed was to reduce a nation-wide mortality (Indians excluded) of 87 in 1921 to 47 in 1941. During this same period bed accommodation of a permanent type increased throughout the Dominion to an average of two per death, and in three provinces to more than three per death.

The outbreak of war again in 1939 led many to believe that this favorable trend would be gravely imperiled, but to a limited degree only have these fears materialized. True, the minimum mortality rate of 1939 has not been quite maintained in 1941 and 1942, but the rise is not great, and determined efforts to control it are evidently already succeeding.

The chief factors causing an actual increase of tuberculosis morbidity are: first, the all-time high employment ratio with the intensity of effort and the long hours in factory work on the part of those thus employed,

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including females at the most susceptible ages; second, the reduced medical and other personnel available for public health work, many sanatorium doctors and nurses having enlisted in the armed forces. The outlook, indeed, would be bad were it not that these adverse factors are being counterbalanced by an intelligent program based primarily on the appreciation of the value of x-ray study of large groups of the population. Since September, 1939, every member of the armed forces of Canada has been x-rayed on enlistment and again when discharged, using a single 14x17 film. Any doubtful film is referred with the patient to a chest consultant. This undertaking has been described by the Secretary of the Canadian Tuberculosis Association as the greatest case finding program Canada has ever had. True it has raised (and corrected) the known morbidity rate but such a rise is temporary and likely to be followed by a permanent drop. Of the first 400,000 volunteers (to December, 1941) one per cent (W. P. Warner) were rejected because of active or potentially active tuberculosis. Practically all of these 3,987 were asymptomatic and apparently healthy, in fact having already passed rigid physical examination. The prompt provision of treatment for these (in the great majority of cases before becoming infectious) reduces to a minimum the exposure to tuberculosis of those in the fighting forces as well as their civilian contacts. Even so, the development of tuberculosis in this well-selected, x-ray negative army overseas is not negligible. Based on the numbers already *invalided* to Canada, the morbidity rate for tuberculosis is 94 per 100,000 per annum and for pleurisy with effusions 70 (Adamson). Clearly such figures emphasize the need of revisional examinations. Valuable as is a single community survey, it is insufficient.

Complementary to the examination of recruits is the mass study by fluorography of those engaged in industry. This, too, is uncovering many unsuspected cases of tuberculosis and removing them from circulation. Such a survey in Ontario of 74,228 persons recently showed 997, or 1.3 per cent, to be tuberculous. Of these, 204, or 0.3 per cent of the total, were considered active or requiring close supervision. These figures, approximating the percentage in recruits, are interesting as suggesting the amount of unrecognized tuberculosis in the general population. The discovery among recruits, soldiers, and in industry of so many more cases of tuberculosis than anticipated by the standards of 1939 has created a problem of finding beds, but, fortunately, this has been minimized by the fact that the average period of treatment required has been short, commensurate with the early stage of the disease at the time of diagnosis. The immediate urgency of the situation has demanded utilization in some degree of temporary accommodation, but long-range plans are being made for the time when priorities will not be the chief governing factor.

Thus, World War II, like its predecessor, is actually accelerating in Canada the favorable trends of 1939 diagnostic and treatment measures. If, with similar enthusiasm after victory, we continue to apply the knowledge we already have, if the programs already undertaken in the name of Total War are, in the Peace, further developed and amplified in the name of Social Security, the day of the irreducible minimum may not be beyond measurable distance. To paraphrase Churchill, We have the tools, let us finish the job.

# Tuberculosis Control in Hawaii

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The Territory of Hawaii consists of seven major inhabited islands forming the eastern end of the Hawaiian Archipelago, and a chain of reefs and small islands forming the western end of the group. The main islands are Hawaii, Maui, Molokai, Lanai, Oahu, Kauai, and Niihau. The estimated population of the Territory in 1941 was about 465,000. In 1941 the city of Honolulu, on Oahu, had a population of 200,000. For the same year, the total population of Oahu was 310,000, of Hawaii 68,000, of Maui (including Molokai and Lanai) 52,000, and of Kauai (including Niihau) 33,000.

The population of the Territory is a mixed one and, as may be imagined, presents interesting problems in tuberculosis epidemiology. The partitioning of the population according to race in 1941 was as follows:

| | | | |
|---|---|---|---|
| Japanese | 159,534 | Chinese | 29,237 |
| Caucasian | 141,627 | Hawaiian | 14,246 |
| Part-Hawaiian | 52,445 | Puerto Rican | 8,460 |
| Filipino | 52,060 | Korean | 6,881 |
| | | All others | 849 |

The population of the Territory, and particularly that of the city of Honolulu, has grown rapidly during the past decade.

The basic industries of the Territory are agricultural, the raising of sugar cane and pineapple. Much stock-raising and considerable small farming is done. Honolulu is a modern, thriving port and city. The military and naval activities centered in the Territory need no mention.

A Board of Health was established in the Hawaiian Islands in 1850 by order of the ruling king, Kamehameha III. It was not, however, until 1900 that tuberculosis was recognized as an important public health problem. In 1910 a Tuberculosis Bureau was established. The director of this bureau is a full-time physician. The Bureau, located in Honolulu, acts in a general supervisory capacity for the control of tuberculosis throughout the Territory. It also conducts diagnostic and follow-up clinics for the island of Oahu as a whole, and discharges the routine statistical duties customarily handled by such a Board of Health department.

Each of the major islands, Oahu, Hawaii, Maui, and Kauai, possesses a tuberculosis sanatorium under a full-time medical director and staff. The largest institution, the Leahi Hospital in Honolulu, is caring for about 450 patients at the present time and has developed an enviable record in thoracic surgery. All of the sanatorium directors are physicians well trained in the specialty of tuberculosis and all carry on an active program of collapse therapy on their respective islands. The sanatorium directors on the islands of Hawaii, Maui, and Kauai act as local representatives of the Tuberculosis Bureau and conduct diagnostic, follow-up, and pneumothorax clinics. Tuberculosis control on all the islands is greatly facilitated by the medical services provided by the plantations. Each plantation has its own hospital and full-time physician. The plantation hospitals are

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well equipped and the physicians are tuberculosis-minded. Patients are x-rayed on the slightest suspicion that they may have a chest lesion. The sanatorium directors give free consultation in all non-pay cases, which means a high proportion of the plantation employees. When it is considered that, except for the island of Oahu, most of the population is made up of plantation employees, the significance of the contribution of plantation medical service to tuberculosis control becomes evident.

There are two tuberculosis associations in the Territory, both affiliated with the National Tuberculosis Association. The Tuberculosis Association of Kauai serves the latter island only, while the Territorial Tuberculosis Association serves all the other islands. Both associations are active organizations.

The Territorial death rate for tuberculosis was 208 in 1901. The death rate in 1941 was 53. Tuberculosis ranked fourth among the ten principal causes of death in the Territory during the fiscal year 1941-1942, the ranking order being as follows: diseases of the heart, accidental deaths, malignant tumors, tuberculosis, congenital malformations and diseases of early infancy, nephritis, intracranial vascular lesions, pneumonia, diabetes mellitus, and suicide.

The following table copied from the Territorial Board of Health report for the fiscal year ending in 1941 gives some idea of the racial distribution of tuberculosis in the Hawaiian Islands:

| *Race* | *New and Re-activated Cases* | *Deaths* | *Rate* | *Avg. Rate 1937-38-39-40-41* |
|---|---|---|---|---|
| Hawaiian | 89 | 34 | 237.71 | 219.75 |
| Part-Hawaiian | 63 | 23 | 44.70 | 69.73 |
| Puerto Rican | 14 | 4 | 47.67 | 38.27 |
| Caucasian | 56 | 16 | 12.82 | 15.64 |
| Chinese | 53 | 24 | 82.66 | 77.59 |
| Japanese | 290 | 79 | 49.76 | 61.71 |
| Korean | 28 | 10 | 145.62 | 135.31 |
| Filipino | 146 | 48 | 91.72 | 111.17 |
| Others | 1 | .. | ..... | ..... |

The average death rate for the Territory for the years 1937 to 1941, was 68.08.

As elsewhere throughout the world, the ratio of minimal to advanced cases diagnosed is unfavorable in the Territory of Hawaii. During 1942 the cases admitted to the sanatorium on Kauai were diagnosed as follows: 16 per cent minimal, 36 per cent moderately advanced, and 48 per cent advanced. On Oahu the hospitalized cases during 1938-1939 were divided as follows: 17 per cent minimal, 25 per cent moderately advanced, and 58 per cent far advanced.

Bovine tuberculosis is an insignificant problem in the Territory.

The present war has produced definite changes in the tuberculosis situation in the Territory. Board of Health figures for the fiscal year ending June 30, 1943, show 1949 cases of active tuberculosis on the register as compared with 1551 at the close of the fiscal year ending June 30, 1942. At the close of the fiscal year 1940 there were 1474 cases on the register. How much of the recent increase is actual and how much is relative to population changes is difficult to estimate at present. Because of the military situation, population statistics have not been released since 1941.

Many factors probably contribute to the increased incidence of tuberculosis in the Territory. Black-out is perhaps one of them. A great many of

the island people, Japanese and Filipinos in particular, live under poor housing conditions even in peace time. Black-out and curfew hours cause these people to congregate in crowded, poorly ventilated quarters, thus greatly favoring the transmission of tuberculosis. Absorption of healthy men into the armed forces, migration of other healthy men to industrial areas (Oahu), and the general opportunity to make good money has caused the weak, the infirm, the aged, and the retired individual to be hired and put to work at unaccustomed occupations. Inability to stand the strain brings them to medical attention and the diagnosis of tuberculosis that otherwise might have remained undiscovered. In other words, strain is bringing to our attention cases which a general survey of the population would have revealed in earlier stages of the disease. Many, if not most, cases of productive tuberculosis go for years without being suspected until discovered accidentally or brought to attention by just some such factor as prolonged strain. In general, the apparent increase in the incidence of tuberculosis in the Territory can perhaps be attributed to two factors, viz.: an increased opportunity for the transmission of the disease due to crowding and poor ventilation, and a lowered resistance of the individual due principally to fatigue resulting from long hours of work, short hours of rest, and the general strain of life under war conditions.

The island of Oahu has apparently been hit the hardest by the change in tuberculosis incidence. This is not because the increase has been anything near epidemic proportions but because hospitalization facilities on Oahu have never been quite adequate, the Leahi Hospital always having had a waiting list. Oahu, of course, is the industrial center of the Territory. There are located the city of Honolulu and the great military and naval bases. How much the population of Oahu has increased since the war began is impossible to state. That the importation of defense workers from the mainland and the migration of other workers from the outlying islands has increased the population is, of course, obvious. It is no military secret that Oahu as a whole and Honolulu in particular have become beehives of activity. The social and economic emergencies accompanying such circumstances are very favorable to the spread of tuberculosis.

It is interesting to note the changes in regard to tuberculosis on the other islands, which do not have large urban centers of population. In a personal communication, Dr. Edmund Tompkins, Director of Kula Sanatorium on Maui, states that he expects the admission rate for Maui for 1943 to be double that of recent years. Dr. W. F. Leslie, Director of Puumaile Hospital on the island of Hawaii, has informed the writer that there is a definite increase in tuberculosis on Hawaii and that the waiting list of the hospital is presenting serious problems. This is very interesting to the writer who is the Director of Mahelona Hospital on Kauai. Kauai is very similar to Maui and Hawaii in quality of population and agricultural pursuits, yet the incidence of tuberculosis on Kauai has scarcely been disturbed by the war. There has been an increase in the number of admissions to Mahelona Hospital during 1943, but nearly all of these excess admissions can be accounted for by deliberate survey activities and hence do not necessarily indicate an actual increase in incidence.

When Territorial population statistics are released at the end of the war, and the facts in regard to the local tuberculosis situation can be correctly analyzed, some very interesting and perhaps valuable information will be available.

# EDITORIAL

## *THE NEED FOR PLANNING THE ECONOMIC REHABILITATION OF THE TUBERCULOUS*

A great deal of thinking and planning is being done regarding post-war physical, mental and economic rehabilitation of men and women in the armed services. To the contrary, little or no planning is taking place for the physically handicapped who are serving on the home front despite their physical limitations.

As is well known, there are arrested, apparently arrested, quiescent, and even mildly active tuberculous individuals who are doing their part by taking on war jobs and working long hours in war industries. Some of these people have already relapsed, others will no doubt follow suit and become active cases, and will transmit their disease to their fellow-workers.

The public health aspect of economic rehabilitation of the tuberculous patient has not been given the consideration that it merits by those who are responsible for the control of tuberculosis. Looking back into the history of the problem, one discovers that official agencies have consistently underestimated the significance of rehabilitation; and even today comparatively few public health officials recognize its importance. Thousands of dollars are spent annually to allay symptoms and to check progress of this disease, yet the importance of appropriating funds for the purpose of safeguarding patients from relapse after discharge from the sanatorium is not appreciated.

The economic status of the majority of tuberculous patients is such that immediately after their discharge from the sanatorium they are prematurely compelled to accept full-time employment, which frequently is unsuited to their work capacity. Inevitably such premature, full-time, unsuitable work promotes a reactivation of the disease, leading to infection to the household and other contacts, thus creating a breach in the defenses against its spread in the community.

Those who are interested in the control of tuberculosis must ultimately realize that the supervision of the tuberculous individual must not end with his discharge from the sanatorium. The great number of relapses and the many deaths which occur within a relatively short time after discharge from the sanatorium make one wonder whether much of the good work of the sanatoria and a considerable amount of public funds are not being wasted.

It should be clearly brought home to the responsible agencies that to prevent infection to the workers in industry, to the community in general, and to reduce mortality, we must prevent relapses; to avoid relapses, it is imperative to provide social and economic security for the tuberculous individual after his discharge from the institution. Such security can in a large measure be achieved if, through the medium of vocational training during convalescence, the patient has been prepared to bridge the gap between the measured pace of sheltered sanatorium life and the accelerated momentum of industrial life.

It is to be hoped that from the many relapses that will unfortunately take place during the period of war, the lesson will be driven home more forcefully that the *economic rehabilitation* of the tuberculous must be a part of any program for the control of tuberculosis. No person can remain physically well if he is not economically rehabilitated to enable him to select the type of work compatible with his physical condition. It should be borne in mind that the economically maladjusted tuberculous individual remains a potential reservoir of infection and therefore a potential menace to the family, the industrial worker and the community at large.

Dr. H. I. Spector, F.C.C.P.

# TEACHING TUBERCULOSIS IN MEDICAL SCHOOLS
## REPORT OF THE
## COUNCIL ON UNDERGRADUATE MEDICAL EDUCATION*

Those of you who are assembled here know that one of the objectives of the American College of Chest Physicians, and to my mind the most important objective as far as the control of tuberculosis is concerned, has been to bring about the establishment in the medical schools of this country an effective method of teaching tuberculosis. A method of teaching whereby the student, when he leaves medical school, will have an *understanding* of tuberculosis that will enable him, no matter what field of medicine he enters, to discharge his responsibility regarding this disease. A responsibility which he should and must discharge if tuberculosis is to be brought under control. The great majority of those leaving medical schools at the present time do not have that understanding. Perhaps the most unfortunate aspect of this situation is that these medical graduates, as they go out as our physicians, for the most part, do not realize that they do not have this understanding and ability.

Medical schools cannot overlook the fact that, in spite of what has been accomplished in the fight against tuberculosis, that this disease is still the most serious medical and health problem with which any state or community has to deal; that tuberculosis is still the leading cause of death in that important group of our population within the age period of from fifteen to forty-five years.

The efficiency of any medical school must be measured as being in direct proportion to the service it renders to the public as a whole. From the point of view of the medical schools, the most serious unsolved problem that confronts the public today is tuberculosis.

It has been my privilege for the past six years to act as Chairman of the Council of this organization in charge of this particular objective. After a number of years of study, going into as much detail as possible as to how this subject was being handled by our different medical schools, this Council formulated a schedule or outline for the teaching of chronic diseases of the lungs. It was our hope that this outline would serve as a guide and that it would be adopted by the medical schools insofar as their particular circumstances would permit.

Two years ago a copy of this schedule, with an explanation of why it was formulated was sent to each medical school in this country. Since that time this Council, through our Chicago office, with the help of the official representatives of the College, in different parts of the country has endeavored to keep in contact with the medical schools relative to this teaching program. In order to bring to you at this time a report of developments that have taken place so far, through the central office of the College two months ago I sent a letter of inquiry to all the medical deans, to the governors of the College, to the presidents of the Chapters and to the members of my Council asking that they report the progress made in their schools or the schools in their vicinity.

There were no follow-ups to these letters sent out and while all the schools did not reply, replies came much easier, showed much more inter-

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*Presented before the meeting of the Board of Governors and Regents, Cincinnati, Ohio, November 17, 1943.

est in the subject and were much more encouraging than were the replies to the first correspondence sent out six years ago. In all, replies were received from over sixty of our medical schools. More than thirty deans or assistant deans wrote personal letters. A fairly large number of replies were received from the heads of the tuberculosis or medical department. In practically all cases a real interest in this work was expressed.

Quite a number who replied stated that because of the manpower shortage incident to the war, they had been unable to take any definite action along this line. Not a few of those replying in this way added the information that they were not satisfied with their present setup for teaching tuberculosis and expressed the hope that after the war definite improvement could be made. Some schools have already arranged their plan of teaching along the lines submitted in the schedule. Other schools, realizing no doubt that, incident to the war, the seriousness of tuberculosis will be increased, are taking definite steps even under present conditions, to improve their setup for teaching this subject. A few schools have recently established a separate department for the teaching of tuberculosis. In the case of a relatively small number of schools the information supplied was not so encouraging. In some instances the subject of tuberculosis is still being presented to the students only through short periods of rather intensive instruction composed of didactic lectures and clinics or clinical clerkships or both, some of these institutions having the added improvement of a few weeks residency for the students in a sanatorium. Under those circumstances, the difficulty of trying to teach a chronic disease as acute diseases are taught is encountered. Tuberculosis is a disease of months and years and since its course is characterized by unforeseen and unpredictable changes, to teach it effectively it must be taught as it occurs. Still, other schools are attempting to teach tuberculosis as a part of the general medical course and in some cases where the instructors are not specializing or are not specially trained in tuberculosis. Again, some schools apparently devote a major part of the time allotted to this subject in attempting to perfect the student in the physical examination of the chest, most of which time could well be spent on other phases of the work. Physical examination of the chest, even in the hands of those who are regarded as experts, as statistics show, does not reveal positive findings in more than one out of four cases of tuberculosis in the early stages, that is, at a time when the diagnosis should and must be made.

Your Council fully realizes that the bringing about of conditions where all the medical schools will present this subject to the student, so that he as a future physician will be able to play the part he must play to bring tuberculosis under control, will be a slow process. They have, however, been more than encouraged by the general interest already shown and the action taken.

Your Council wishes to express its appreciation to those members of the College who have been so helpful in carrying out this work and to our Chicago office which has made the work possible. The Council also wishes to stress the fact that if this objective is to be accomplished it can only be accomplished by the united and continued effort of the general membership and particularly of you who are its official representatives.

Dr. E. W. Hayes, F.C.C.P.
*Chairman*

# COLLEGE NEWS

*Program*

## NORTH MID-WEST REGIONAL MEETING

*American College of Chest Physicians*

The members of the American College of Chest Physicians in the states of Iowa, Minnesota, Nebraska, the Dakotas, and Wisconsin, which comprise the North Mid-West Regional District, will meet at Rochester, Minnesota, Saturday, April 15, 1944.

This meeting will be held in conjunction with the 91st annual meeting of the Minnesota State Medical Association, at Rochester, Minnesota, April 13-15. An invitation has been extended by the Minnesota State Medical Association to the members of the American College of Chest Physicians to present a program on diseases of the chest and the following program has been prepared for this meeting:

*Program on Diseases of the Chest*

The Arena, Rochester, Minnesota

Saturday Morning, April 15, 9:00 A. M.

"Practical Points in the Diagnosis of Pulmonary Tuberculosis"—Dr. S. A. Slater, F.C.C.P., Medical Director, Southwestern Minnesota Sanatorium, Worthington, Minnesota.

"Development of Therapy in Tuberculosis During the Last Twenty-five Years"—Dr. John F. Allen, F.C.C.P., Omaha, Nebraska.

"Present Status of Chemotherapy in Tuberculosis"—Dr. H. C. Hinshaw, Mayo Clinic, Rochester, Minnesota.

" 'All-Out' Tuberculosis Control by the Medical Profession"—Dr. Karl Danielson, Meeker County, Litchfield, Minnesota.

"Sarcoidosis"—Dr. W. L. Meyer, Medical Director, South Dakota State Sanatorium, Sanator, South Dakota.

Case Reports—Dr. L. W. Moody, Medical Director, Pureair Sanatorium, Bayfield, Wisconsin.

*Luncheon*

Carlton Hotel, Rochester, Minnesota

"Transitory, Migratory Pulmonary Infiltrations Associated with Eosinophilia"—Dr. J. Winthrop Peabody, F.C.C.P., President, American College of Chest Physicians, Washington, D. C.

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It is important that members of the College who plan on attending this meeting should communicate *at once* with Dr. A. M. Olson, Chairman, Room Reservations Committee, Mayo Clinic, Rochester, Minnesota.

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Dr. Karl H. Pfuetze, F.C.C.P., Cannon Falls, Minnesota, Chairman, General Arrangements Committee.

Dr. Jay Arthur Myers, F.C.C.P., Minneapolis, Minnesota, Chairman, Program Committee.

*Governor's Committee*

Dr. Gustav A. Hedberg, F.C.C.P., Nopeming, Governor for Minnesota, Chairman.

Dr. John F. Allen, F.C.C.P., Omaha, Governor for Nebraska.

Dr. J. Carl Painter, F.C.C.P., Dubuque, Governor for Iowa.

Dr. Carl O. Schaefer, F.C.C.P., Racine, Governor for Wisconsin.

Dr. A. L. Banyai, F.C.C.P., Wauwatosa, Regent.

For further information regarding this meeting, please communicate with Dr. Karl H. Pfuetze, F.C.C.P., Medical Director, Mineral Springs Sanatorium, Cannon Falls, Minnesota.

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## CHAPTER MEETINGS

*California Chapter*

The California Chapter, American College of Chest Physicians, will hold its annual meeting at the Hotel Biltmore, Los Angeles, California, May 7, 1944. This meeting will be held in conjunction with the annual meeting of the California Medical Association, which meets at Los Angeles, May 7-8, 1944.

For further information regarding this meeting, please communicate with Dr. J. J. Singer, Secretary-Treasurer, California Chapter, American College of Chest Physicians, 2200 West Third St., Los Angeles, California.

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*Illinois Chapter*

The annual meeting of the Illinois Chapter, American College of Chest Physicians, will be held at the Palmer House, Chicago, on May 16, 1944. The Illinois Chapter of the College will meet with the Illinois State Medical Society holding its annual meeting at Chicago, May 16-18.

Colonel Arnold Dwight Tuttle, Medical Director, American Airlines, will be the guest speaker at a luncheon meeting of the Chapter and he will present a paper on the "Effect of Air Travel on Cardio-respiratory Diseases." The annual business meeting, reports of the committees and election of officers will follow the luncheon meeting.

For further information concerning this meeting, please communicate with Dr. Julius B. Novak, Secretary-Treasurer, Illinois Chapter, American College of Chest Physicians, 2800 Milwaukee Avenue, Chicago, Illinois.

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*Joint Meeting—Indiana and Kentucky*

The Kentucky members of the American College of Chest Physicians have invited the members of the Indiana Chapter of the American College of Chest Physicians to meet with them at Louisville, Kentucky, on Sunday, April 16. A scientific program is being prepared for this meeting and the guest speaker will be Dr. J. Winthrop Peabody, President, American College of Chest Physicians, Washington, D. C.

For further particulars concerning this meeting please communicate with Dr. Paul A. Turner, F.C.C.P., Regent of the College, Superintendent and Medical Director, State Tuberculosis Sanatorium, Louisville, Kentucky.

### *Missouri Chapter*

The Missouri Chapter, American College of Chest Physicians, will hold its annual meeting at the Muehlebach Hotel, Kansas City, Missouri, April 24-25 in connection with the annual meeting of the Missouri State Medical Association.

For further information concerning this meeting, please communicate with Dr. Melvin Tess, Secretary-Treasurer, Missouri Chapter, American College of Chest Physicians, 3611 St. Louis Avenue, St. Louis, Missouri.

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### *New Jersey Chapter*

The New Jersey Chapter, American College of Chest Physicians, will hold a luncheon meeting at the Hotel Claridge, Atlantic City, New Jersey, April 26, 1944, in connection with the annual meeting of the Medical Society of New Jersey. A business meeting will follow the luncheon. Reports of the officers and committees, a review of progress during the past year, and plans for the further progress of the chapter will be made.

For further information concerning this meeting, please communicate with Dr. Irving Willner, F.C.C.P., Secretary-Treasurer, New Jersey Chapter, American College of Chest Physicians, 18 Waverly Avenue, Newark, New Jersey.

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The New Jersey Chapter, American College of Chest Physicians, has been invited by the New Jersey Tuberculosis League to send two or more delegates to a joint conference on Social Hygiene and Tuberculosis to be held at the Essex House, Newark, New Jersey, April 20, 1944.

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### *New York State Chapter*

The New York State Chapter, American College of Chest Physicians, will hold its annual meeting at the Hotel Pennsylvania, New York City, Thursday, May 11, 1944. This meeting will be held in conjunction with the annual meeting of the New York State Medical Society, which meets at New York City, May 8-11, 1944. Officers will be elected for the coming year, and other chapter business will be discussed at the meeting.

For further information concerning this meeting, please communicate with Dr. Arthur Q. Penta, President, New York State Chapter, American College of Chest Physicians, 713 Union Street, Schenectady, New York.

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### *Ohio Chapter*

The Ohio Chapter, American College of Chest Physicians, will hold a luncheon meeting at the Deshler-Wallick Hotel, Columbus, Ohio, on May 3, 1944, in connection with the annual meeting of the Ohio State Medical Society. The following program has been arranged for the meeting:

1) Treatment of Tuberculous Empyema.
2) Question Box.
3) X-Ray Conference.

For further particulars concerning this meeting, please communicate with Dr. Myron D. Miller, F.C.C.P., President, Ohio Chapter, American College of Chest Physicians, Superintendent and Medical Director, Franklin County Tuberculosis Hospital, Columbus, Ohio.

*Texas Chapter*

The Texas Chapter, American College of Chest Physicians, will hold its annual meeting at the Texas State Tuberculosis Sanatorium, Sanatorium, Texas, April 23, 1944. A scientific program will be presented at this meeting and plans for post-war tuberculosis teaching will be discussed.

For further information concerning this meeting, please communicate with Dr. Elliott Mendenhall, Secretary-Treasurer, Texas Chapter, American College of Chest Physicians, 1217 Medical Arts Building, Dallas, Texas.

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## COLLEGE NEWS NOTES

*A Letter from a Fellow Overseas*

"I want to let you know that I appreciate receiving literature regarding our organization, and the excellent publication, *Diseases of the Chest*. The subject matter and method of presentation of the articles in the magazine give me much enjoyment and a chance to keep in touch with my specialty while in Army service. That is sincerely appreciated."

Captain Garry G. Bassett, M.C., 29th Field Hospital, APO 730, c/o Postmaster, Seattle, Washington.

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Dr. S. Aubrey Gittens, F.C.C.P., New York City, Associate Physician, Harlem Hospital, has been appointed Tuberculosis Clinician with the New York City Department of Health, in charge of Tuberculosis at the Fort Greene Health Center, Brooklyn, New York.

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Dr. Evarts A. Graham, F.C.C.P., St. Louis, Missouri, was awarded the Charles Mickle Fellowship for 1943 by the University of Toronto Faculty of Medicine, for his work in "The Diagnosis and Treatment of Carcinoma of the Lung." On November 8, Dr. Graham was granted honorary Fellowship in the Royal College of Surgeons of England at a ceremony held in the British Embassy at Washington, D. C.

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Dr. Maurice G. Buckles, F.C.C.P., Louisville, Kentucky, was elected to the office of President-Elect of the Jefferson County Medical Society.

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Dr. Herman E. Hilleboe, F.C.C.P., Senior Surgeon Medical Officer in Charge of Tuberculosis Control Section, U. S. Public Health Service, Washington, D. C., presented a paper before the Annual Congress on Industrial Health of the American Medical Association, at the Palmer House, Chicago, Illinois, February 15, 1944.

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Dr. Chevalier L. Jackson, F.C.C.P., Philadelphia, Pennsylvania, was a guest speaker at the Eighth Annual Session of the New Orleans Graduate Medical Assembly held at New Orleans, Louisiana, March 6-9.

Dr. James L. Bibb, F.C.C.P., Chattanooga, Tennessee, addressed the Chattanooga and Hamilton County Medical Society on March 2. Dr. Bibb spoke on "Some New Methods of Handling the Tuberculosis Problem."

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Dr. C. Alvin Dougan, F.C.C.P., Honolulu, Hawaii, has resigned as Director of the Tuberculosis Bureau of the Territory of Hawaii Board of Health. He is now situated at Kahuku, T. H.

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"California and Western Medicine" publishes a release from the "Associated Press" to the effect that Dr. Paul C. Samson, F.C.C.P., Oakland, California, performed a total pneumonectomy on a badly wounded German soldier, under adverse conditions, in a Station Hospital at Algiers, Africa. The Allied Headquarters Surgeon's Office reports that the patient is alive and well.

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Dr. William J. Grosfeld, F.C.C.P., Paterson, New Jersey, was appointed Medical Director of the Morgan County Tuberculosis Sanatorium, Decatur, Alabama.

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## REVIEW

### *SOVIET MEDICINE*

*Diseases of the Chest* hails with enthusiasm a new medical journal, the *American Review of Soviet Medicine*, published by the American-Soviet Medical Society. This society was founded in the Spring of 1943 to promote cooperation between the members of the medical and allied professions of the U. S. A. and the U. S. S. R., and to collect and exchange information about medical developments and achievements in the medical and allied sciences of the two countries. The need for such an organization is recognized by all physicians, and the distinguished leadership of Dr. Walter B. Cannon and Dr. Leo Eloesser is assurance of success.

*The Review* is edited by Dr. Henry E. Sigerist. The ninety-four pages of the first number are divided among main articles, clinical notes, and survey articles translated from the Russian, a profile of Nikolai N. Burdenko, an editorial analysis of health work in the Soviet Union, reviews of Soviet books, and society news and notes. The tone and format are impressive. At a time when many a publication is struggling to exist it is stimulating to see one born so auspiciously.

Sheldon Domm, M.D.

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## PRELIMINARY PROGRAM

*Tenth Annual Meeting*

## AMERICAN COLLEGE OF CHEST PHYSICIANS

Stevens Hotel, Chicago, Illinois—June 10-12, 1944

### *Symposium on Tracheo-Bronchial Tuberculosis*

Radiological Aspects—Dr. C. C. Birkelo, Detroit, Michigan.

Pathological Aspects—Dr. Gertrude A. Silverman, New York, N. Y., and Dr. Moses J. Stone, F.C.C.P., Boston, Massachusetts.

Surgical Aspects—Dr. Richard H. Overholt, F.C.C.P., Brookline, Massachusetts.

Clinical-Medical Aspects—Dr. H. McLeod Riggins, New York, N. Y., and Dr. Moses J. Stone, F.C.C.P., Boston, Massachusetts.

### *Collapse Therapy and Surgery of the Chest*

Artificial Pneumothorax Among Negro Patients—Dr. Reuben Hoffman, F.C.C.P., Maryland Tuberculosis Sanatorium, Henryton, Maryland.

Mechanical Paralysis of the Left Hemidiaphragm Complicating Collapse Therapy in Pulmonary Tuberculosis—Dr. Oscar Feinsilver, F.C.C.P., Worcester, Massachusetts.

Various Types of Thoracic Operations—Dr. Ralph C. Matson, F.C.C.P., Editor, *Diseases of the Chest*, Portland, Oregon.

Segments of the Lung from the Standpoint of Surgical Procedure—Major Brian B. Blades, M.C., Walter Reed General Hospital, Washington, D. C.

Chronic Pleural Empyema, Its Surgical Treatment—Lt. Col. John B. Grow, MC, F.C.C.P., Fitzsimmons General Hospital, Denver, Colorado.

### *Tuberculosis in Institutions*

Tuberculosis in Prisons—Dr. Otto L. Bettag, F.C.C.P., Tuberculosis Controller, Illinois State Penitentiary, Pontiac, Illinois.

Tuberculosis in Institutions for the Mentally Ill—Dr. H. A. Burns, F.C.C.P., Division of Public Institutions, St. Paul, Minnesota.

Tuberculosis in American Colleges and Universities—Dr. Ruth E. Boynton, Director, Student Health Service, University of Minnesota, Minneapolis, Minnesota.

Tuberculosis Control in American Schools—Dr. Chester A. Stewart, Director, Department of Pediatrics, School of Medicine, Louisiana State University, New Orleans, Louisiana.

Tuberculosis Control in Industry—Dr. Herman E. Hilleboe, F.C.C.P., Senior Surgeon Medical Officer in Charge Tuberculosis Control Service, U. S. Public Health Service, Washington, D. C.

### *Pneumonia and Allergy*

Pitfalls in the Diagnosis of Atypical Pneumonia—Major Walter L. Nalls, M.C., F.C.C.P., Walter Reed General Hospital, Washington, D. C.

Irreversible Allergy in Nontuberculous Diseases of the Chest—Dr. Milton B. Cohen, F.C.C.P., Cleveland, Ohio.

### *Chemotherapy in Tuberculosis*

Present Status of Chemotherapy in Tuberculosis—Dr. H. C. Hinshaw and Dr. William H. Feldman, Mayo Clinic, Rochester, Minnesota.

Diasone in the Treatment of Pulmonary Tuberculosis (One Year's Experience)—Dr. Karl H. Pfeutze, F.C.C.P., Cannon Falls, Minnesota.

*X-Ray*

Roentgenography of the Soft Tissues of the Thoracic Cavity and Neck—Dr. Jose de Carvajal-Forero, Warner Institute for Therapeutic Research, New York, N. Y.

The Value of Film Files of Radiographs of the Chests on Miniature Film for Direct Use with Viewers as a Teaching Aid—Major Linneus G. Idstrom, M.C., Station Hospital, Camp Crowder, Missouri, and Dr. H. L. Hiebert, F.C.C.P., Kansas State Department of Health, Topeka, Kansas.

*Other Subjects*

Patent Ductus Arteriosus—Dr. James W. Nixon, F.C.C.P., San Antonio, Texas.

Hedblooms Syndrome—Dr. Minas Joannides, F.C.C.P., Chicago, Illinois.

The Care of Tuberculous Veterans—Colonel Roy A. Wolford, MC, F.C.C.P., Assistant Medical Director, Tuberculosis Division, U. S. Veterans Administration, Washington, D. C.

Acute Diaphragmitis (Hedbloom's Syndrome)—Dr. Minas Joannides, F.C.C.P., Chicago, Illinois.

*Speakers from Other Countries*

Atelectasis, Pneumonitis, or a similar subject to be presented by Lt. Col. J. D. Adamson, R.C.A.M.C., Department of National Defense, Ottawa, Canada.

Diabetes in Tuberculosis to be presented by a delegate from the Brazilian Chapter of the American College of Chest Physicians.

Pneumonitis—Lt. Col. J. D. Adamson, R.C.A.M.C., Department of National Defense, Winnipeg, Canada.

Papers for the meeting are also being prepared by Dr. Donato G. Alarcon, President, Mexican Chapter, American College of Chest Physicians, by Dr. Ovidio Garcia-Rosell, Lima, Peru, Governor of the College for Peru, and by members of the other Pan-American Chapters of the College.

*Refresher Course in Diseases of the Chest*
*To Be Presented by the*
*Illinois Chapter, American College of Chest Physicians*

The Present Status of Tuberculosis in Children—Dr. Arthur S. Webb, F.C.C.P., Tuberculosis Controller, DuPage County, Glen Ellyn, Illinois.

Management of Minimal Tuberculosis—Dr. Julius B. Novak, F.C.C.P., Medical Director, Tuberculosis Institute of Chicago and Cook County, Chicago, Illinois.

Chemotherapy in Tuberculosis—Dr. Charles K. Petter, Medical Director and Superintendent, Lake County Tuberculosis Sanatorium, Waukegan, Illinois.

The Treatment of Bronchiectasis—Dr. Edwin R. Levine, F.C.C.P., Medical Director, Chest Service, Michael Reese Hospital, and The Winfield Sanatorium, Chicago, Illinois.

Bronchoscopic Aspects of Bronchial Tuberculosis and Recent Advances in the Technic of Bronchoscopy—Dr. Paul H. Holinger, F.C.C.P., Assistant Professor, Department of Otology, Rhinology, and Laryngology, University of Illinois, College of Medicine, Chicago, Illinois.

Indications for Collapse Therapy—Dr. Richard Davison, F.C.C.P., Tho-

racic Surgeon, Municipal Tuberculosis Sanitarium, Associate Professor of Surgery, School of Medicine, Loyola University, Chicago, Illinois.

Surgical Procedures in Tuberculosis—Dr. Jerome R. Head, F.C.C.P., Attending Surgeon, Edward Hines, Jr., Hospital, Assistant Professor, Thoracic Surgery, Northwestern University Medical School, Chicago, Illinois.

Surgical Procedures in Non-Tuberculous Disease—Dr. Ralph Bettman, Associate Professor of Surgery, University of Illinois, College of Medicine, Chicago, Illinois.

Pregnancy in Tuberculosis—Dr. Fred M. F. Meixner, F.C.C.P., Chief, Diseases of Chest Section, Methodist Hospital, Peoria, Illinois.

*Note:* A number of other prominent physicians will present interesting papers and their names and the titles of the papers will be published in the next issue of our journal, which will contain the complete program for the meeting.

### *"Information Please"*

The usual interesting and informative program which has appeared on College programs since 1940 and which is known to our members as "Information Please" in medicine, will again be presented at this meeting. Experts on diseases of the chest, thoracic surgery, bronchoscopy, roentgenology, and pathology will answer your questions. These questions will be directed by the Moderator to these experts and they will be answered spontaneously and without rehearsal.

### *Council on Military Affairs and Public Health*

The Council on Military Affairs and Public Health is preparing a program which will bring to the members of the College the latest developments in tuberculosis and other diseases of the chest in World War II. Officials from the Surgeon General's Office of the U. S. Army, Navy, Public Health Service, and Veterans' Administration have been invited to appear on this program.

### *Conference of Chapter Officials*

The Annual Conference of College Chapter Officials will be held in conjunction with this meeting. All chapter officials should arrange to attend this conference.

### *Other Sessions*

The Executive Council, Board of Regents, and Board of Governors will meet to transact the business of the College, and officers of the College whose terms of office have expired in 1943 and those which will expire in 1944 will come up for election. Recommendations for all elective offices should be sent to the Nominating Committee:

Dr. E. W. Hayes, F.C.C.P., Monrovia, California, Chairman; Dr. Robert K. Campbell, F.C.C.P., Springfield, Illinois, and Dr. Francis J. Welch, F.C.C.P., Portland, Maine.

### *Board of Examiners*

The next written examination for Fellowship in the American College of Chest Physicians will be given by the Board of Examiners of the College at Chicago, June 9, 1944. Candidates for Fellowship in the College, who desire to take the examination should communicate with the Executive Secretary of the College as early as possible.

# OBITUARIES

## WOODBURN J. HUDSON

*1886-1944*

Dr. Woodburn J. Hudson died in the Hahnemann Hospital, Philadelphia, Pennsylvania, on January 18, 1944. Dr. Hudson was born in Camden, New Jersey, the son of Eli and Mary Hudson. He graduated from the Hahnemann Medical College, class of 1910. He served his internship at the Reading Hospital, Reading, Pennsylvania. In 1911, he opened offices for the practice of medicine at Pleasantville, New Jersey, where he remained until his death.

For the past eighteen years, Dr. Hudson served as Medical Director of the Pine Rest Tuberculosis Sanatorium. During World War I, Dr. Hudson held at the rank of 1st Lieutenant in the Medical Corps.

He was a member of the Atlantic City Hospital staff, New Jersey State Homeopathic Society, American Medical Association, and a Fellow of the American College of Chest Physicians.

Dr. Hudson was active in civic and fraternal organizations in Pleasantville and throughout Atlantic County. He was past commander of the Mainland American Legion Post No. 81, past president of the Kiwanis Club, a member of the Keystone Lodge F. and A. M., Crescent Temple, Excelsior Consistery, and the Pleasantville Lodge No. 213, I.O.O.F. He became a member of the Pleasantville Fire Department in 1916, and was elected chief in 1920, serving in that capacity until he resigned last year.

Dr. Hudson is survived by his wife, Jessie; a son, Major Howard Hudson; a sister, Mrs. Elwood Horan; and a brother, Russell Hudson, of Atlantic City.

Dr. Marcus W. Newcomb, F.C.C.P.
Governor of New Jersey

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## FRANK HALL WASHBURN

*1871-1943*

Frank H. Washburn, governor of the American College of Chest Physicians for Massachusetts, died November 10, 1943, in the New England Baptist Hospital, Boston, Massachusetts. He was born at Falmouth, Maine, September 21, 1871, the son of Lorenzo and Maria Barker Washburn, descendants of John Washburn who settled in Duxbury, Massachusetts, about 1630. He was educated in the public schools of Falmouth, Maine, attended Westbrook Seminary, Deering, Maine, and Shaw's Business College in Portland, Maine. He attended the Massachusetts College of Pharmacy in Boston and registered in 1893 as a pharmacist. He graduated from Tufts College Medical School, Boston, in 1899; served his internship at Lynn Hospital, Lynn, Massachusetts, from 1899 to 1900 and started his medical practice in Jamaica Plain, Massachusetts. Shortly after that, he developed tuberculosis and went to the Rutland State Sanatorium for treatment. He again commenced practicing medicine in Jefferson, Massachusetts, about 1902, later moving to Holden where he continued practice until his death.

During the early years, he traveled by horse and buggy far and wide, in good and bad weather, ministering to the sick. At that time he did considerable major surgery on the kitchen table in the home, having in mind the idea of a hospital at some future time. About 1914 the Holden Cottage Hospital was started at the home of Mr. and Mrs. George Jenkins on Pleasant Street where Dr. Washburn and the local physicians comprised the staff. This hospital soon became inadequate and in November, 1922, the present Holden District Hospital was opened. Dr. Washburn became surgeon and chief of staff, which position he held until his death.

In 1919 the Holden Clinic was started, Dr. Washburn being the director and sole owner until September, 1942, at which time a partnership of practicing physicians of Holden was formed to perpetuate the clinic. Besides being surgeon and chief of staff at the Holden District Hospital, Dr. Washburn was thoracic surgeon at the Worcester County Sanatorium and consulting surgeon at the Rutland State Sanatorium; Harrington Memorial Hospital, Southbridge; Webster District Hospital and consulting urologist at the U. S. Veterans Facility at Rutland Heights, Massachusetts. He was governor of the American College of Chest Physicians since its inception for the state of Massachusetts. He was director of the Rutland Training Center and a member of their board of directors. He served as a member of the Procurement and Assignment Service for this district. He was a member of many medical societies among them the Worcester District Medical Society of which he was a past president and a counselor and member of the Board of Censors for many years. He was a Fellow of the American College of Surgeons, and of the American College of Chest Physicians; a member of the American Urological Society, New England Roentgen Ray Society, New England Trudeau Society, American Trudeau Society and a charter member of the Wachusett Medical Improvement Society. He was a member of many fraternal and sportsmen organizations, among them the Holden Trowel Club, Boylston Lodge, A. F. & A. M. Worcester County Commandery, Worcester Royal Arch Chapter and Alethia Grotto. From the earliest days of his medical career, he submitted articles to the leading medical journals of the country. These were always well prepared and were received with the greatest of interest by his colleagues.

Dr. Washburn devoted all of his energies to the medical profession which he dearly loved. He was untiring in his efforts to bring solace to the people who came to him for relief of illness. He was never satisfied with mediocrity and was always trying for perfection in his work. In fact, he instilled in his associates this love of perfection and he was always urging others to attain higher ideals and better results. His professional ethics were something beautiful to observe and know. They were above reproach, and often he was the one to be grieved or to suffer because of his high professional standard of ethics.

Dr. Washburn remained youthful in his medical knowledge and work. He kept abreast of the times and often somewhat ahead of the latest medical authorities. His advice was sought by many and he was always ready to lend a helping hand to his colleagues. He was especially keen to help young associates in their early struggles to start practice.

In his passing, we, who knew him best, have suffered a severe blow. His memory will never be erased from our minds.

Dr. Moses J. Stone, F.C.C.P.
Regent, New England States

## JOHN LUTHER REEVES

*1900-1943*

John Luther Reeves was born at Washington, D. C., February 5, 1900. He attended the public schools in the District of Columbia, and graduated in medicine from Howard University in 1923. He interned at the City Hospital, Kansas City, Missouri, and established a private practice in Philadelphia in 1925.

For eight years he conducted the City Chest Clinic at the Phibbs Institute and later was chest physician at the City Clinics of St. Lukes and Rush Hospitals.

Dr. Reeves devoted his life to the service and wellbeing of his fellowmen. He was married to the former Elsie Brooks, and they have one son, John Luther.

John H. Bisbing, M.D., F.C.C.P.
Governor for Pennsylvania

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## BOOK REVIEW

*Surgery of the Heart.* King, E. S. J., M.D., M.S., D.Sc., (Melb.), F.R.C.S. (Eng.), F.R.A.C.S., Major A.A.M.C. 728 pages, 276 illustrations. First edition, 1941. Price: $13.50. Williams and Wilkins Co., Baltimore.

This monograph is based on the Jacksonian Essay of the Royal College of Surgeons for 1938 and consists of observations made on pathological material, experimental work, clinical cases, and wide use of the literature.

The text is divided into two main sections. Section one (182 pages) consists of a consideration of the anatomy, histology, physiology, etc., of the heart together with a brief review of the main features of electrocardiographic and radiological methods of study of cardiac conditions. Section two deals with the treatment of cardiac disease and is divided into three parts: (1) the general methods of surgical approach and the various principles on which they are based (48 pages); (2) the experimental work which has been done to elucidate the various problems (14 pages); and (3) the various diseases and their treatment (436 pages).

The book therefore is by no means merely a manual of operative surgery. Rather its chief value is that of a truly comprehensive appraisal of the literature on practically all phases of cardiology from the viewpoint of a surgeon. The author's attitude throughout is critical and sound but always optimistic regarding the relatively unexplored regions of cardiac surgery.

Unfortunately the book is no longer up to date. For example the discussion of ligation of patent ductus arteriosus is considered worthy of only one paragraph, the significant sentence of which states: "In many cases there are other significant abnormalities, and even if these could be excluded it is extremely probable that the sudden alterations in the circulation induced by the ligation of such a relatively large vessel would have the most serious and immediate consequences." p. 277.

The sections on injuries of the heart are excellent, as is the discussion of the surgery of the pericardium including pericardiectomy in Pick's disease. The evaluation of the Beck-O'Shaughnessy procedures for the establishment of artificial collateral circulation is critical. The author prefers the production of pericardial adhesions by means of the insuffla-

tion of sterile bone dust into the pericardial cavity under local anesthesia.

This book will be of great value to the specialist in cardiac surgery, especially to one interested in research, because of the admirable summary of the literature, the many suggestions for future investigation, and the insistence on adherence to basic principles and controlled series. The book is of less value to the physician whose prime interest is not the surgery of the heart but who wishes to keep informed on the current status of the subject.

Sheldon Domm, M.D.

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## ABSTRACT

*Clinical Concept of Hemoptysis, Use of Liver Extract*; *Dr. A. Pareja* Coronel, *Anales de la Sociedad Medico Quirurgica del Guayas*, Ecuador.

The author points out that fulminating hemoptysis does not always originate from rupture of large vessels. Some of these hemorrhages are due to pathological changes on the walls of the vessels with disturbance of their vasomotor and vasotrophic properties resulting probably from disorders of hepatic function. In three cases of fatal hemoptysis associated with tuberculosis, the author found at necropsy little or no evidence of hemorrhage in the areas of the lung most involved by the tuberculous process; in contrast with that in the non-tuberculous areas, there was congestion, hemorrhage and escape of bloody fluid from the bronchi. Because of the important role of the liver in the production of the factors involved in the coagulation of blood, the author felt that the use of liver extract might be beneficial in the control of hemoptysis. He tried it in over fifty patients. His results were satisfactory in patients in whom other measures had failed to control the hemorrhage.

Dr. L. M. Tocantins

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